VETERANS ADMINISTRATION HOSPITAL ISSUE

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VOLUME 29 MARCH, 1957 NUMBER 3

PREVENTION OF DEFORMITIES

SYMPOSIUM:

INTESTINAL OBSTRUCTION AND BLEEDING

Complete Contents on Page iv

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REFERENCES

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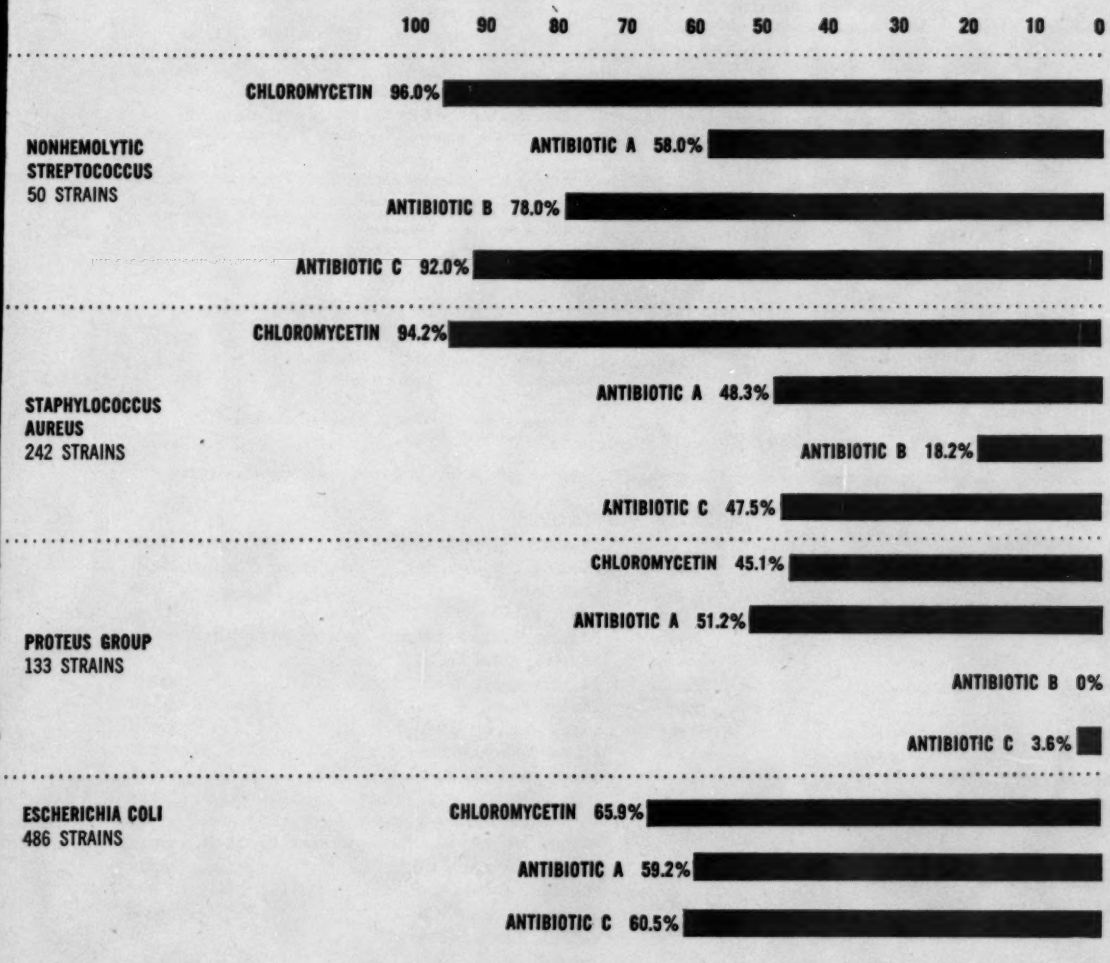

*This graph is adapted from Rantz and Rantz.[4] It is based on *in vitro* studies of bacteria freshly isolated from clinical materials.

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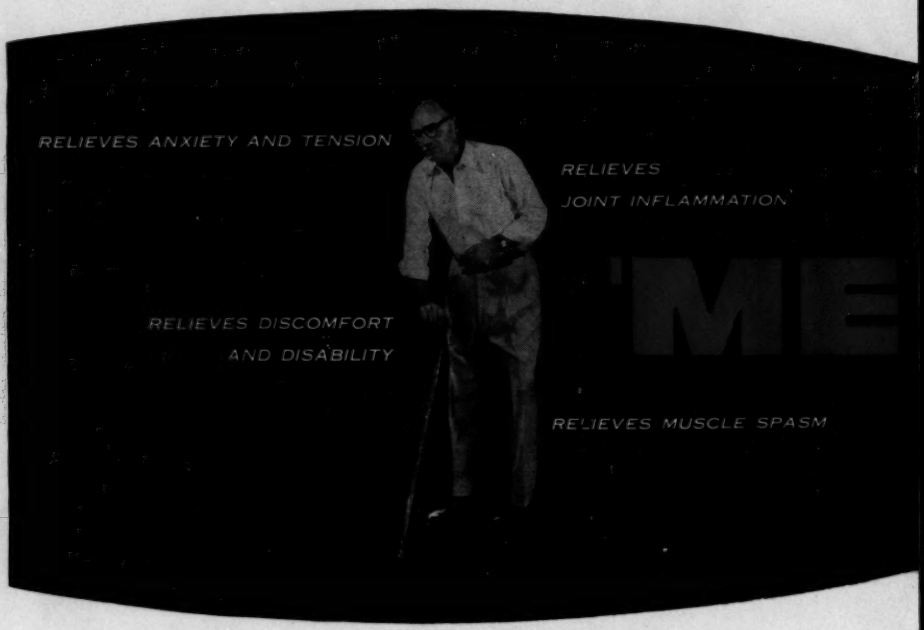

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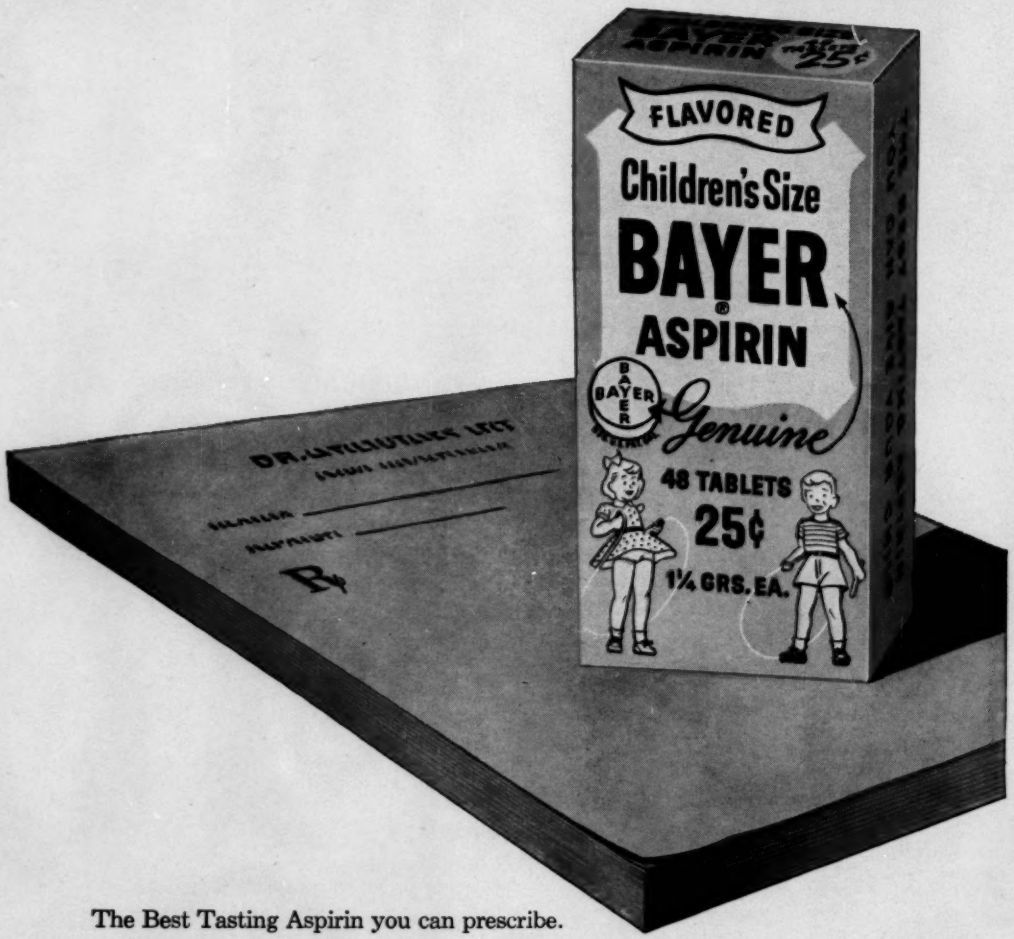

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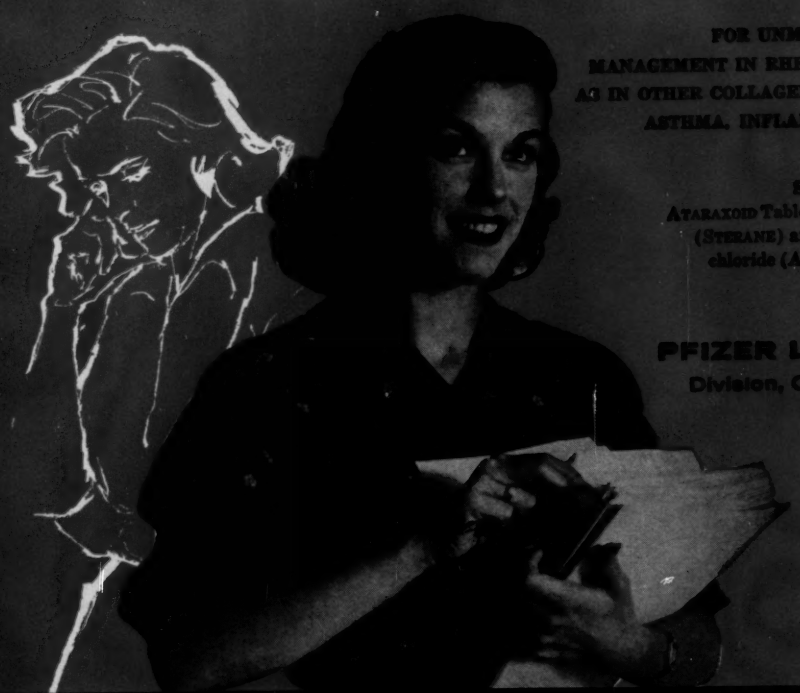

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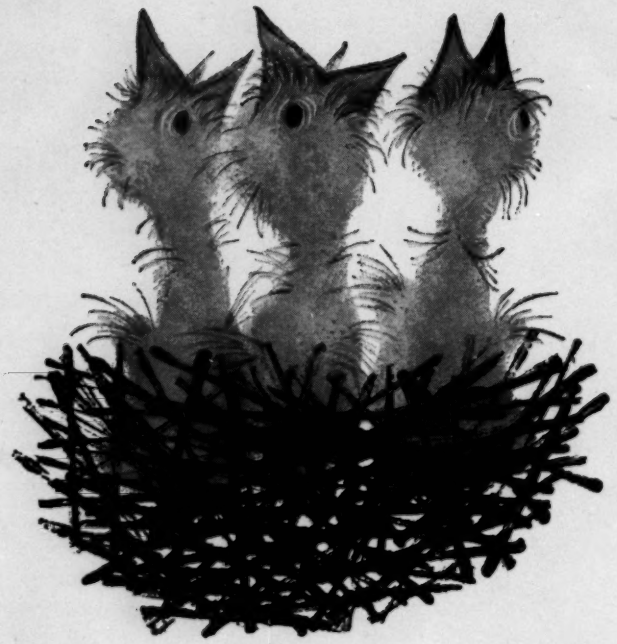

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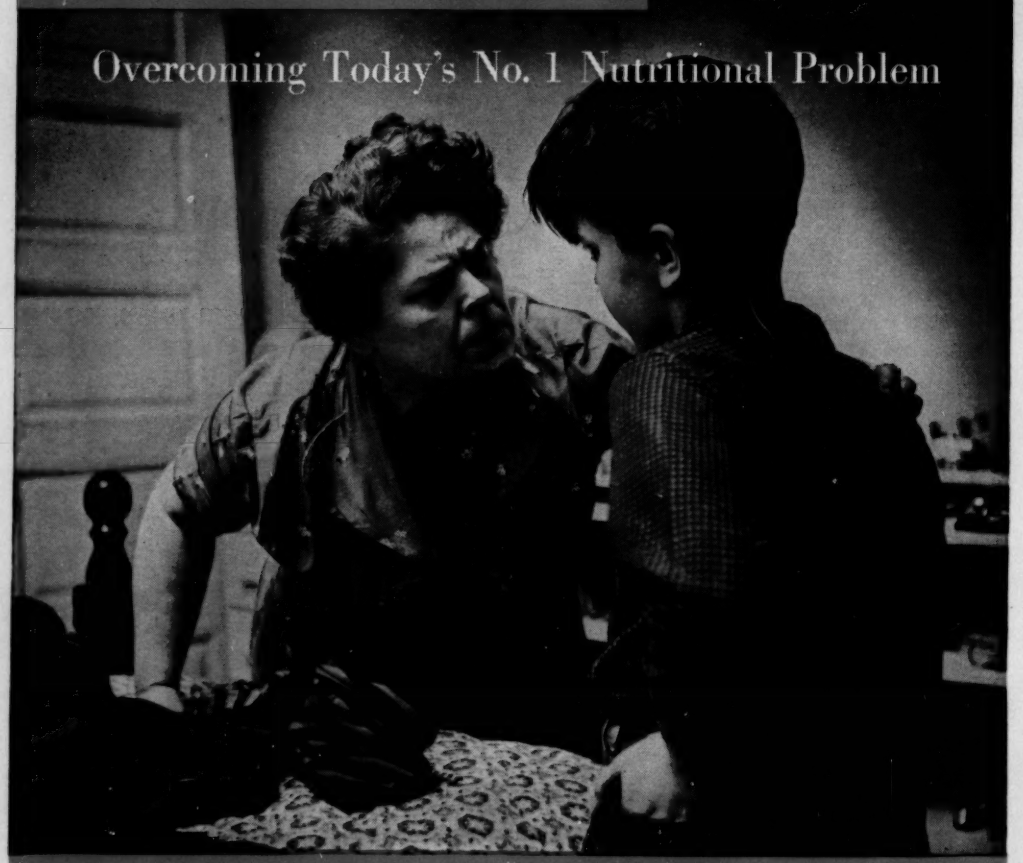

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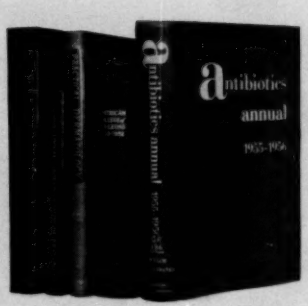

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2. Waddington, W. S., Maple, F. C., and Kirby, W. M. M., A.M.A. Archives of Internal Medicine, 1954, p. 556.

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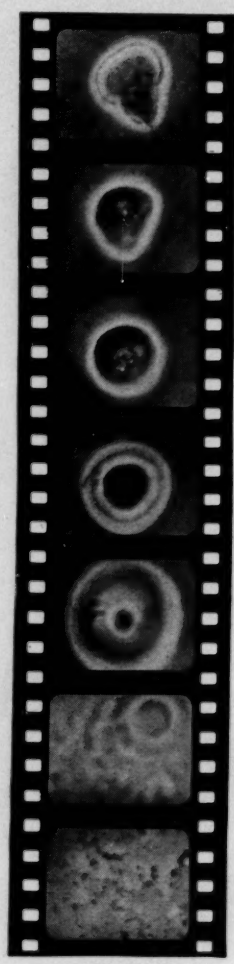

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15 sec. EXPLODES
16 sec. SCATTERS

References: 1. Davis, C. H.: J.A.M.A. *157*:126 (Jan. 8) 1955. 2. Davis, C. H.: West. J. Surg. *63*:53 (Feb.) 1955. 3. Davis, C. H., and Grand, C. G.: Am. J. Obst. & Gynec. *68*:559 (Aug.) 1954. 4. Davis, C. H. (Ed.): Gynecology and Obstetrics (revision), Hagerstown, Md., W. F. Prior, 1955, vol. 3, chap. 7, pp. 23-33. 5. Draper, J. W.: Internat. Rec. Med. *168*:563 (Sept.) 1955.

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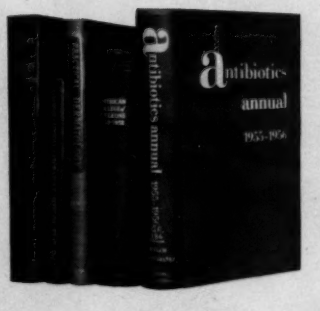

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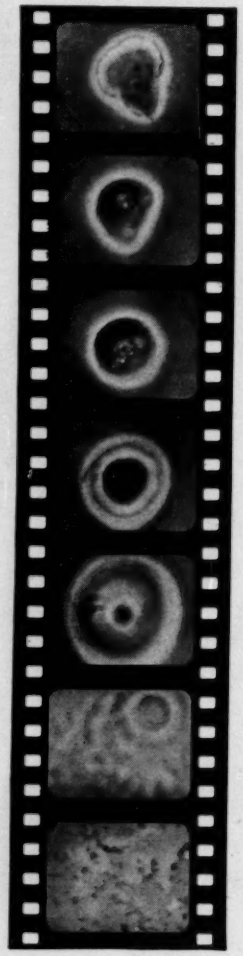

Top to bottom:
2 sec. CONTACTS
4 sec. COMPLEXES
6 sec. DISSOLVES
8 sec. DENATURES
10 sec. SWELLS
15 sec. EXPLODES
16 sec. SCATTERS

WITH THE Davis technique,† using VAGISEC® liquid and jelly, flare-ups of vaginal trichomoniasis rarely occur. VAGISEC liquid actually explodes trichomonads within 15 seconds after douche contact.[1] Better than 90 per cent apparent cures follow use of this new trichomonacide,[2] developed as "Carlendacide" by Dr. Carl Henry Davis, noted gynecologist and author, and C. G. Grand, research physiologist.[3]

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HOME TREATMENT—*Patient douches with* VAGISEC *liquid every night or morning and then inserts* VAGISEC *jelly. Home treatment is continued through two menstrual periods, but omitted on office treatment days. Douching is contraindicated in pregnancy.*

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References: 1. Davis, C. H.: J.A.M.A. *157*:126 (Jan. 8) 1955. 2. Davis, C. H.: West. J. Surg. *63*:53 (Feb.) 1955. 3. Davis, C. H., and Grand, C. G.: Am. J. Obst. & Gynec. *68*:559 (Aug.) 1954. 4. Davis, C. H. (Ed.): Gynecology and Obstetrics (revision), Hagerstown, Md., W. F. Prior, 1955, vol. 3, chap. 7, pp. 23-33. 5. Draper, J. W.: Internat. Rec. Med. *168*:563 (Sept.) 1955.

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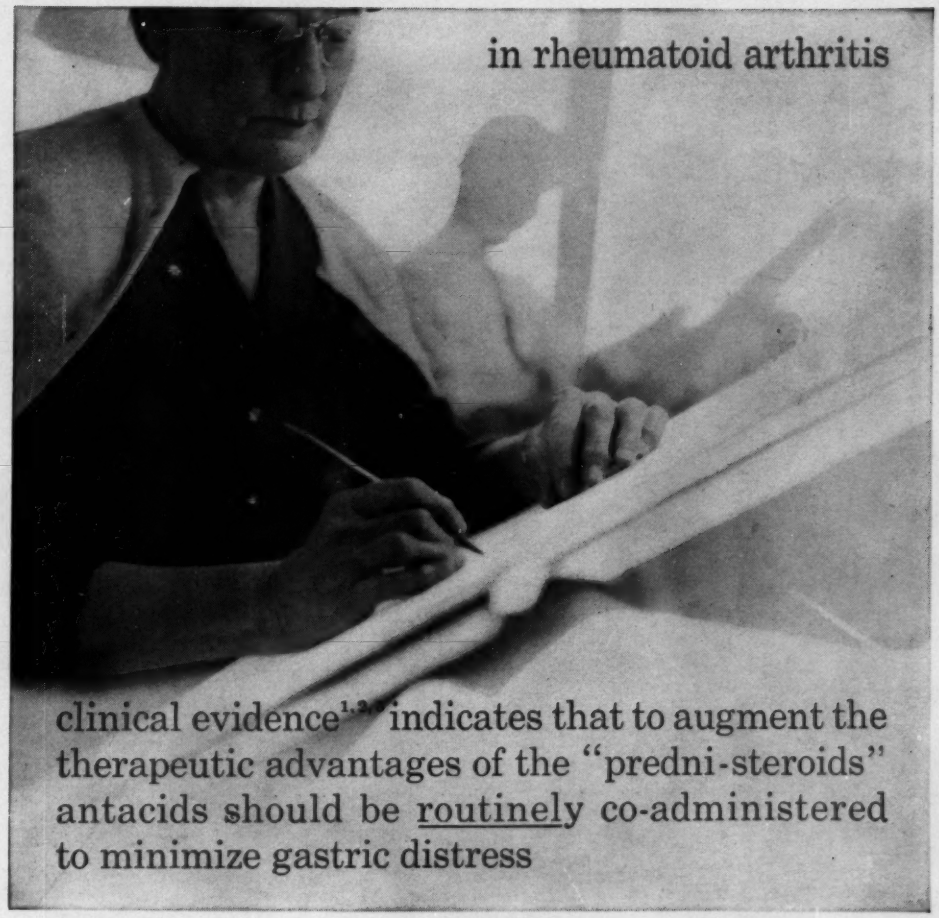
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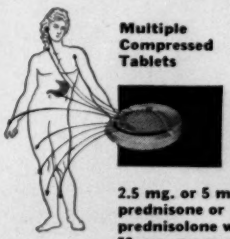

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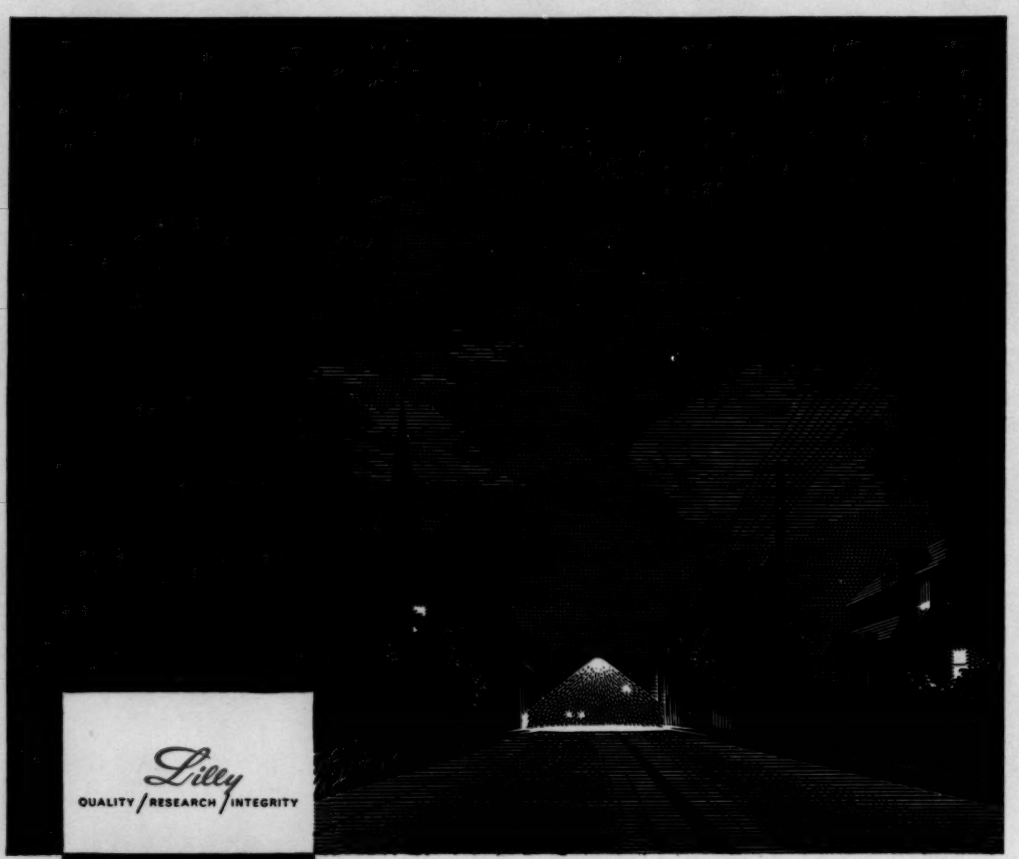

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DEFORMITIES CAN BE PREVENTED

FREDERICK E. DUGDALE, M.D.C.M.*

One of the changes brought about by growth and development in medical knowledge is an increase in the length of the human life span. Because of a longer life expectancy and advance in technological skill, additional problems are created. Machines are the sources of increased crippling disabilities, and chronic diseases are causes of physical disabilities in an aging population.

The general practitioner is the first to see, recognize, and treat many of the chronic diseases. Therefore, my purpose is to point out some preventable after effects of chronic diseases and to discuss procedures by which the family can assist in their prevention. All but the most serious patients should be treated in the home as many elderly persons are easily projected into a psychotic state by institutionalization. The family and physician have available in the community a valuable ally to assist them in many of these procedures. She is the visiting nurse.

A common affliction of the older age group (and frequently of younger individuals also) is the cerebral vascular accident. Among the residual disabilities of the hemiplegic patient are the painful "frozen shoulder" and the flexion contractures of the upper extremity. These conditions may be prevented in most cases with ten minutes of daily attention.

Early consideration must be given to the bed position of patients for the period of acute shock. A comfortable position may be obtained by placing a pillow in the axilla of the affected side with the arm across it so that the shoulder is fully abducted to 90°. The forearm should lie across the pillow, the elbow flexed 90° and the arm internally rotated, the palm flat and the fingers fully extended. On four occasions daily the hand and forearm should be picked up and the arm fully externally rotated. The palm will be facing up. The procedure should be reversed to return the arm to the original position. If this maneuver is repeated from six to eight times on each occasion it will keep a shoulder free and "frozen shoulder" can be prevented. At the same time it is easy to flex and extend the elbow fully and to move the fingers through a full range of motion.

The affected lower extremity should be braced by a small pillow on the lateral side beneath the greater trochanter to prevent external rotation of the leg and foot. A few minutes spent three or four times daily to flex the thigh on the abdomen and the knee on the thigh six or eight times will maintain joint motion in this extremity. If the patient can at any time voluntarily lift the heel from the bed with the leg straight, he should be able to walk.

Barring complicating factors of cardiac or other disease, prolonged bed rest is to be deplored in the treatment of the hemiplegic patient. Active treatment and ambulation should be started as soon as the period of acute shock has passed. The incontinence of bowel and bladder should respond rapidly and without further attention to early ambulation. It will assist further in the maintenance of protein metabolism, with calcium and phosphorous balance, to prevent muscle wasting, urinary calculi, and osteoporosis. The threat and consequences of decubiti also are avoided.

One of the greatest crippling diseases is rheumatoid arthritis. It frequently leaves

* Chief Physical Medicine and Rehabilitation Service, Veterans Administration Hospital, Wilmington.

the patient deformed due to preventable flexion contractures. The patient soon learns that the pain in his knee is eased by placing a pillow beneath the joint. This malposition brings about knee flexion contractures. The hip flexors shorten, producing a combined knee-hip flexion contracture. Ambulation with such position is impossible. In fact, attempted ambulation under such circumstances further aggrevates the problem of walking.

Other symptoms of the arthritic patient are upper extremity deformities: the "tight" shoulder, flexed elbow, wrist and hand deformities with the fingers flexed, subluxed and ulnar deviated. Back weakness may occur if the spine is involved. Therefore, the combination of poor positioning, muscular weakness and atrophy, result in deformities. A patient in this condition requires long term physical restorative procedures, and the end results may leave much to be desired.

For the general practitioner to outline a routine for the arthritic patient takes perhaps ten to fifteen minutes; but the family, by following these simple instructions can save a patient much time, financial loss, and suffering.

To help prevent such consequences some simple measures are suggested. A board should be placed beneath the mattress. The head and shoulders should not be raised on pillows to flex the upper spine. Proper alignment of the joints in the acute stage of the disease insures functional body mechanics when activity is feasible. The upper extremity should be placed to avoid the malpositions previously described. With the upper, as with the lower extremity, half-shell, padded plaster splints are an aid. The limbs should be removed from such casts three or four times daily, and all joints should be put through a full range of motion within the limits of pain. Analgesics before such motion makes the program easier for both the patient and his attendant.

"Muscle setting" should be done hourly by the patient when awake. This simple maneuver prevents or slows muscle atrophy and weakness, and improves the circulation of the involved periarticular structures and musculature. The patient is instructed to contract specific muscle groups, without joint motion, and to hold the contracture from five to ten seconds before relaxing it.

The lower extremities must be kept in extension when not being exercised. I prefer the previously mentioned padded plaster shell from mid-thigh to beyond the toes, with the foot at right angles for prevention of heel cord shortening and foot deformities. A pillow doubled and placed against a foot board in the bed can be used also to give added support to the feet.

Most deformities seen in arthritic patients, with the possible exception of those of the hand, are due to extensor weakness. The extensors are stretched and weakened and the flexors are allowed to shorten and contract. Therefore, the proper body positioning and maintenance of joint range of motion are basic principles in the treatment of arthritic and hemiplegic patients. Unless the physician keeps them in mind, an otherwise sound medical program may fail.

In the discussion on arthritis the use of a pillow under the knee was condemned. The same principle applies to patients with diabetes, arteriosclerosis, or other peripheral vascular diseases. The same knee and hip flexion contractures may be intiated. If gangrene follows, requiring amputation, postoperative progress may be impaired. The use of pillows under a stump for more than 24 hours invites a flexion contracture which complicates the satisfactory adaptation to a prosthesis. When amputation becomes inevitable the patient should be physically and psychologically conditioned for the postoperative course by ancillary assistance from the hospital's department of physical medicine to strengthen the upper extremities for crutch walking pre-and post-amputation. Early post-surgical stump exercise is indicated and will decrease pain and improve healing.

What physician would send his patient to a pharmacist with instructions to get some medication for a cold? Serious thought should also be given to the prescription of a prosthesis. A patient who has had a re-

cent amputation is poorly prepared to be referred to a limb-maker without a specific prosthetic prescription. Few amputees are capable of adequate adjustment to and use of a prosthesis without some instructive assistance. If the physician is too busy to undertake the supervision of such a program he should assist the patient to obtain the necessary training.

With the increasing aging population more crippling disabilities can be anticipated. Some conditions which can be prevented have been presented.

With the knowledge, tools, and technics at our disposal, many permanent disabilities can be either minimized or wholly avoided. Granting the apparent inevitability of varying degrees of crippling due to the nature of the basic pathology, we must recognize the frequency of superimposed secondary pathologic changes that can be avoided by the meticulous observance of intelligently planned preventive measures. The statement by Hippocrates, "Do your patient no harm," has greater implication today than when it was made.

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INTESTINAL OBSTRUCTION

DONALD G. MCHALE, M.D.*

A definite decrease in the mortality rate of patients treated for intestinal obstruction has been noted in the past several decades. This improvement in treatment testifies to the effort, experimental work, time, study and consideration given to it by numerous investigators. A working knowledge of the physiological, pathological and biochemical aspects of this problem is essential if one is to treat these cases on anything other than an empirical basis.

Late cases of intestinal obstruction that have been successfully treated by the "suction method" (i.e. Miller-Abbott, Levine and Cantor tubes, etc.) have shown that "fatal toxemia" is not of necessity a constant accompaniment of a high grade obstruction of the intestine. The success of suction treatment also favors the mechanistic and bacterial explanations of the cause of death. This theory is that death is due to the loss of viability of the gut wall and subsequent permeation of bacteria and other products. It appears that the utilization of the Cantor and Miller-Abbott tubes have relegated the "toxemic effect" to the end of the process in causing its added lethal effects.

Perhaps the simplist way to consider intestinal obstruction is to use a pathological classification which divides all obstructions into:

I. SIMPLE OBSTRUCTION in which the continuity of the bowel alone is blocked and where there is no primary vascular disturbance; e.g., adhesive bands. Further subdivision could be incomplete and complete. Two entities, due to nervous inbalance, may be included in simple obstruction; viz., paralytic or inhibition obstruction and spastic or dynamic obstruction.

II. STRANGULATED OBSTRUCTION where, in addition to blocking in bowel continuity, the blood supply of the bowel is compromised.

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Previous to any changes caused by obstruction, the bowel has a certain normal enteric pressure with a variable amount of gas, fluid and/or solids. Gases commonly present in intestinal meteorism are nitrogen 75%, oxygen, carbon dioxide, hydrogen, methane and hydrogen sulfide. The proportions are subject to considerable variation. Swallowed air in obstruction represents about 68%; about 22% are gases diffused from the blood, especially nitrogen, and the remainder 10% are due to food fermentation.

The general sequence of events in a typical case of complete simple obstruction of the bowel can be fairly well depicted.

At first with obstruction there occurs an increase in the activity of the bowel. The exact mechanism of this is difficult to explain. With increasing distension and intraluminal pressure there is thinning of the bowel wall and decreased absorption from the intestinal lumen. As the intraluminal pressure increases the diameter of the gut is stretched, thereby jeopardizing first the capillary circulation then the venous and then the arterial channels.

With encroachment on the blood supply, loss of plasma follows injury and later even whole blood can escape. Then follows anoxia, infarction, necrosis and gangrene which allows migration of noxious bacteria and substances through the damaged bowel wall and ultimately transperitoneal absorption. The importance of the bacterial phase of toxicity in strangulating obstructions must be emphasized. In the small bowel a certain amount of decompression occurs by regurgitation of contents back into the stomach, usually with vomiting. In the large bowel either a closed loop occurs or there is regurgitation into the ileum and small bowel. The former happens when the ileocecal valve is competent which it is in about 60% of the cases.

Distension is probably the main factor in the advent of fluid and electrolyte loss

into the "3rd compartment." It also hinders return venous flow and thereby lessens the general effective circulatory volume. Upward pressure on the diaphragms hinders respiration and pulmonary complications occur in proportion to the amount of mechanical and reflex interference.

FLUID ALTERATIONS:

Various investigators have attempted to estimate the rate of digestive fluid "turnover" in 24 hours. This figure could involve 6½ liters or more. A fair proportion of our "mobile internal environment of intercellular fluid" can play a role in bowel obstruction. The loss of acid or base can be fairly well determined depending on whether the obstruction is "high" or "low." In recent years levels of Na, Ka and Cl can be followed and are an aid in our fluid electrolyte replacement program. Some writers stress the amount of blood loss which can occur, especially in strangulating types of obstruction and particularly where the venous obstruction factor predominates.

SYMPTOMS AND DIAGNOSIS:

Having in mind the physiologic, pathologic and biochemical changes, we can consider the symptoms and the diagnosis:

Early in the course of obstruction the patient may not appear to be sick. Pain, as in all abdominal conditions, is of paramount importance. Pain associated with noisy peristalsis should immediately raise the question of intestinal obstruction. Once the suspicion of intestinal obstruction arises, x-rays usually furnish the remaining information. At times the scout film can solve the entire problem. On other occasions, all that can be told is that there is obstruction. We do not use cleansing enemas either as treatment or as a diagnostic maneuver. The barium enema often makes the definite diagnosis.

Palpation of the abdomen may reveal tenderness, peritoneal irritability, its point of maximum intensity and perhaps a mass. This applies usually to the case of strangulated intestine. Associated tachycardia, elevation of temperature and increasing white count (even after restoration of fluid balance) are warnings to delay operation no longer. One of the more difficult diagnostic problems is to differentiate a strangulated bowel from an inflammatory condition complicated by a simple obstruction. Preliminary Cantor tube suction may help solve this situation. I suspect that the early operator has been surprised on occasion at the pathology unearthed by the laparotomy. One recent case of ours seemed quite typical of acute, simple, intestinal obstruction. Preliminary suction was started and laparotomy performed. Many loops of small bowel and the colon were quite severely distended but no obstructive lesion was found. Pancreatic, biliary, renal and other reflex mechanism, we thought, had been ruled out. A trivial back strain was the only event that could be unearthed on repeated close questioning. This had occurred several days previous to what was interpreted as the onset of the patient's complaints and illness. It seemed unlikely to be the etiological factor. If this was a case of reflex ileus it was an unusual one. He recovered uneventfully. One does not often encounter such a situation.

The fundamental concept in treatment is the release of the causative agent before the necessity arises of having to deal with nonviable bowel.

The necessity of restoring fluid-electrolyte balance preoperatively is essential. Preoperative restoration to as nearly normal balance is the goal. This includes blood, plasma, vitamins, amino-acids, in addition to the water, salt, potassium and other electrolytes available today. In addition, early administration of antibiotics is customary.

Concomittantly, intestinal decompression is started. We have not been successful in getting the balloon of the suction tube, whatever the type, past the pylorus in many of the late, distended cases. Cecostomy, or proximal transverse colon colostomy, as early as feasible has been used in the acute colonic obstruction with a closed loop effect.

Prolonged small bowel intubation may appreciably delay progression of the ob-

structive phenomena even with persistence of the causative agent. In some patients it was necessary to delay operation until well into the second week. These patients showed no ill effects of the delay.

We have seen most of the complications arising from the use of decompression tubes. No critical problems were caused by knotting of the tube in the stomach, spillage of mercury or rupture of the balloon. At times when it has been impossible to pass the tube further into the small bowel we have instilled 10 to 30 cc. of lipiodol or thin barium sulphate solution into the tube in an attempt to delineate the obstacle. These maneuvers probably added little toward arriving at a diagnosis.

An adequate vertical incision is usually used at operation. The presence of sanguineous peritoneal fluid is evidence of strangulation. In large bowel obstruction the most distal portion of the colon that appears distended on the x-ray film should be examined. In small bowel obstruction a dilated loop of bowel frequently will present itself. Following the bowel in the direction that presents itself least readily may lead to the obstruction. Usually the small bowel distal to the occlusion will be collapsed. Despite the various suggestions as to bowel recognition (Monk's Localization and Wilms' Suggestions) we have frequently had to find the ileocecal juncture and then trace back the small bowel toward the jejunum. Wangensteen's suggestion of aseptic intestinal decompression by trocar and covering a F-30 rectal tube with a lubricated-on-the-inside Penrose drain has been used with some satisfaction in diminishing soilage. We have not had many cases lending themselves to irrigation with neomycin solutions at time of operation and have no opinion as to its possible value. On several occasions it seemed vital to manually thread the mercury filled balloon down through distended loops, decompressing them en route. This has been performed in association with enterotomy. While soilage was kept to a minimum, the handling of the distended bowel was considerable and would seem too injurious except in certain critical situations. In certain areas where serosa is stripped and dense adhesions must be separated from lengths of small intestine, plication both at the serosa and by suturing the mesentery has been found effective. This is especially valuable in the chronically recurring obstructions of patients previously subjected to multiple abdominal operations.

In conclusion it appears that factors in improving the over-all mortality rate in intestinal obstruction are:

(1) Close attention to restoration of metabolic balance preoperatively.

(2) Adequate antibiotics. Their successful use in the last fifteen years have attested to the importance of the bacterial phase of toxicity in cases of strangulation obstruction.

(3) Limiting unsuccessful decompression to 24 hours.

(4) Earlier diagnosis and operation of strangulated obstructions.

MANAGEMENT OF ACUTE GASTROINTESTINAL HEMORRHAGE

K. SACHS, M.D.*

Gastrointestinal hemorrhage constitutes a major medical emergency in which decisions based on sound medical judgment must be made promptly to prevent loss of life. The majority of patients who have bled from the gastrointestinal tract, especially those in the age group below 40, will recover regardless of the treatment employed. In a considerable number of cases, however, survival may be achieved only by the best of management. This includes personal observations at frequent intervals by the responsible physician.

History and physical examination, which always should include a rectal examination to verify the presence of melena, will usually result in a definite or presumptive diagnosis. Statistically, the bleeding results from peptic ulcer in about 70% of the cases from esophageal varices in 10 to 20% and less frequently from gastrointestinal tumors, hemorrhagic gastritis, blood dyscrasias, and other causes. Passage of a tube into the stomach, the old string test and/or a BSP test may help to determine the site bleeding. However, even in a case of well established Laennec's cirrhosis, the bleeding may still originate from a peptic ulcer or hemorrhagic gastritis. These conditions are found more frequently among cirrhotics than among the general population. The risk of an early x-ray examination of the stomach is a small one and well worth taking in cases where the bleeding is persisting and its origin has remained obscure. In at least 10% of the cases extensive clinical studies fail to find the cause of the bleeding.

TRANSFUSIONS OF BLOOD

The replacement of blood to restore circulating blood volume is an established and essential part of therapy of gastrintestinal bleeding regardless of its origin. The fear that a transfusion of blood will raise the blood pressure and force out a clot is not based on facts except in cases of over-transfusion. In bleeding of short duration the red blood count or hemoglobin determination may not reflect the magnitude of the blood loss. Furthermore, vomiting and/or lack of fluid intake may cause dehydration and a deceptively high hemoglobin level. In cases of rapid blood loss the reduction of circulating blood volume is usually expressed by manifestations of circulatory insufficiency such as tachycardia, tachypnea, pallor, episodes of fainting and decrease of blood pressure. Such patients in whom the circulation is unadapted to the lessened blood volume may need blood very rapidly. Emergency transfusions with unmatched type "O" blood, plasma expanders, or saline solution may be necessary to prevent irreversible shock. Otherwise intravenous saline solution is rarely indicated except in the presence of prolonged vomiting and dehydration. The amount of blood and the speed with which it is given must be adequate to restore and maintain circulation. It is attempted to keep the pulse, blood pressure and respiration at a normal level. In the absence of shock it is preferable to give transfusions of blood at a slow rate to avoid rapid overloading of the circulation and possible undue increase of blood pressure. It is questionable if moderate lowering of the hemoglobin level without shock or syncope should be treated by transfusions but one certainly should not wait for shock to occur. Some writers[1] believe that a systolic blood pressure of 90-100 or lower or a hemoglobin of 70% (10.5 grams) or below are indications for blood transfusion. This is an over simplification and cannot replace clinical judgment. The incidence of homologous serum jaundice in transfusion is 2%. This is a serious complication with a high mortality rate. Other transfusion reactions are not uncommon. Therefore, transfusions should be used when indicated but never needlessly.

BLEEDING FROM ESOPHAGEAL VARICES

Control of bleeding from esophageal varices by insertion of a Sengstaken or

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similar triple-lumen double-balloon tube should be attempted without delay. The swallowing of the tube may be facilitated by the simultaneous drinking of a few sips of water. After insertion of the tube the gastric balloon is inflated with 150-300cc of air. The tube is then partially withdrawn until resistance is felt as the balloon engages at the cardia. The method of taping the tube to the nose and face is not effective to maintain pressure at the gastric side of the cardia and a one pound traction should be applied to the tube by a string running over a pulley at the foot of the bed. Next, the esophageal balloon is inflated to a pressure of 20 to 30mm. of mercury which is usually sufficient to stop the bleeding. It is important that the pressure be maintained in both balloons as an insufficiently inflated upper balloon is worse than useless. Deflation of the lower balloon leads to regurgitation of the tube and obstruction of the airways with disastrous consequences. Pressure in the esophageal balloon can be maintained by a two bottle device working on hydrostatic principle.[2] Next the stomach is aspirated and emptied through the tube and, if there is no further evidence of bleeding, feeding through the same tube may be started. If the tube continues to yield blood after emptying of the stomach and in spite of proper placement and adequate pressure of the esophalgeal balloon, the patient obviously is bleeding from a source below the esophagus and other measures must be taken. Gradual deflation of the esophageal balloon should be attempted after 12 to 24 hours to forestall mediastinitis. Unfortunately, the bleeding frequently recurs following removal of the tube and an attempt may then be made to ligate the bleeding vein. This may be done through the esphagoscope or, more effectively, by way of thoracotomy. At a later time a more definite procedure to lower the increased portal pressure (one of the shunting procedures) may be indicated. The prognosis of those who withstand this procedure is far better than those who are managed conservatively.

In addition to the above the management of a patient who had been bleeding from esophageal varices should be the same as that for a patient with peptic ulcer. One should attempt to neutralize the acid gastric juice by frequent feedings of milk keeping in mind the influence of gastric activity on the causation or recurrence of this type of bleeding.

MANAGEMENT OF BLEEDING PEPTIC ULCER

The mortality is high, at least 10%, if operations are performed on all patients during the early stage of severe bleeding from peptic ulcers. The mortality is less than 1% if operation is delayed to an interval free of bleeding. On the other hand, if an operation is delayed in a patient with continuous bleeding and episodes of shock and anemia but eventually must be performed during active bleeding the average mortality is 25% or more. These figures, agreed upon by most authorities, clearly establish the doctrine that the initial management of patients with bleeding peptic ulcer should be medical. Those few patients who do not stop bleeding under such treatment should be recognized early and operated upon. This can be done only by close observation of the patient during the first 48 hours. This has been termed the period of decision. The decision for an operation should be made sooner in patients over 45 years of age in whom the more rigid arteries make a spontaneous cessation of the bleeding less likely. Recurrent bleeding under adequate medical treatment calls for the same measures as persistent bleeding.

Close cooperation of the internist and the surgeon is required especially during the initial period of decision. Nothing should be given by mouth as long as the patient is vomiting. Morphine should not be used. Strict bed rest and adequate sedation will usually control vomiting, nausea and restlessness. If hematemesis should continue it may be necessary to speed up the transfusion of blood and an early operation should be considered. Most authorities believe that intubation and gastric suction should not be used. The presence of the tube and suction stimulate the stomach to increased peristalsis which is undesirable and removal of the gastric juice may rapidly lead to fluid and electrolyte imbalance. It

is occasionally difficult to determine whether the patient is continuing to bleed. Continued bleeding of significance will manifest itself by hematemesis, melena, or instability of the circulation as expressed by fall of blood pressure, increase of pulse rate, rate of respiration, pallor, increased perspiration or syncope. Therefore, close observation of the patient and interpretation of these vital signs by the physician are imperative. As indicated before, a tube may be inserted to obtain evidence for or against continued bleeding.

Physiologic reasoning and experience of groups in this country and abroad clearly indicate that treatment by starvation to "rest" the stomach leads to poor results and is therefore undesirable. As soon as nausea and vomiting have subsided feedings of milk, or milk and cream, should be started at hourly intervals in amounts of 2 ounces. If well tolerated these may be increased to 4 ounces after 12 to 24 hours. The results of this type of management seem to be superior to the immediate institution of a free feeding or Meulengracht type regimen. However, in a week or so after cessation of the bleeding it makes little difference if a Meulengracht diet or the more gradual increase of a modified Sippy regimen is used. I prefer the latter. *It is of greater importance that the neutralization of gastric acidity not be neglected at any time.* Milk is an excellent antacid. It is unnecessary in most cases that any other antacid be used while the patient is on hourly milk feedings. If, however, the patient is known to have gastric hypersecretion or if the ulcer pain persists after the hemorrhage, an antacid should be used. Calcium carbonate is preferred over one of the aluminum hydroxide preparations because the latter tend to cake when mixed with blood. Bed rest, sodium luminal and milk or antacid usually control the pain of peptic ulcer promptly and effectively. If pain should persist, pyloric obstruction or a penetrating ulcer with impending perforation should be suspected. In this event, which is not very common in bleeding ulcer, gastric aspiration or intermittent suction should be used and fluids and electrolytes lost must be properly compensated by parenteral replacement.

USE OF DRUGS

The usefulness of sodium phenobarbital in the treatment of bleeding ulcer has already been mentioned. It may also be used in Laennec's cirrhosis since it is mainly excreted through the kidneys and there is no significant hepatotoxic effect. However, part of it is degraded in the liver and this phase is impaired in the presence of hepatic damage. Therefore the dosage should be lower and more widely spaced if this condition is present.

Morphine is contraindicated in bleeding peptic ulcer or in esophageal varices because it frequently causes nausea, vomiting, increase of gastric peristalsis, pylorospasm and it may mask an impending perforation. It also depends on the liver for its degradation. Demerol may be used in small amounts. Most authorities advise against the use of atropine in the early posthemorrhagic period because it increases the pulse rate. However, it may be useful to control pain due to increased peristalsis, spasm, nausea and vomiting. This drug or tincture of belladonna is traditionally employed in the treatment of peptic ulcer when bleeding is not a problem or several days after bleeding has stopped. Tincture of belladonna is effective only if used in adequate amounts which are just below the dosage which produces dryness of mouth and blurred vision. Other newer drugs, especially banthine, are useful but there is no evidence that they are in any way superior to atropine preparations.

Vitamin K should be used routinely in bleeding esophageal varices due to Laennec's cirrhosis. Other vitamins are indicated if there is a history or evidence of deficiency or malnutrition or if a prolonged maintenance on a much restricted diet is contemplated. They may be given parenterally during the early posthemorrhagic period.

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BLOOD TRANSFUSIONS

C. DAVIS BELCHER, M.D.*

The use of blood transfusions has increased at such a rapid rate that we now take them for granted. The tremendous boon to mankind represented by the establishment of blood transfusions on a scientific basis cannot be denied. So great and rapid has been our acceptance of blood transfusions that there is a tendency to believe that the procedure is without hazard. This is not true. Furthermore, as blood is transfused on a wider scale with each passing year the inherent dangers may increase. I will discuss briefly the history of blood transfusions and point out where these hazards may arise both now and in the future.

Blood transfusions were attempted in ancient times but many reports are confused. There was no recognition of the closed circulatory system and consequently, in most of the old experiments, there is reason to believe that the blood was swallowed and not actually transfused into a blood vessel. Many magical and potent qualities were attributed to the blood of young people and animals and the ingestion of blood and later its actual transfusion were employed to combat the debility of age and the emaciation of chronic illness. Egyptian princes bathed in blood; in Rome the blood of dying gladiators was deemed particularly suitable for ingestion.

One of the earliest recorded efforts was in connection with Pope Innocent VIII in 1492. Three young men were used as donors and all succumbed, probably from exsanguination. It is believed that the blood was ingested and not transfused.

In the study of the circulation. Galen held the view of a venous blood flowing to and fro carrying nutritive substances from the liver and bowel and an arterial circulation carrying vital spirits and involving the lungs. Michael Servetus in 1953 pointed out the true nature of the pulmonary circulation, but his early death prevented him from completing the picture. Caesalpinus, around 1590, explained the function of the heart as a pump and its method of filling and emptying. Hieronymus Fabricus focussed attention on the valves in the veins, but failed to appreciate their true function. However, his student William Harvey had these facts before him. Prior to Harvey's precise and admirable description of the circulation, Andreas Libavius of Halle in 1615 described a system of direct transfusion which sounds almost modern. In his description he connected artery to artery by a system of tubes. Whether he ever did this and whether he tried to use arteries in both donor and recipient is not known. It seems more likely he was just theorizing, but he recognized a circulatory system and the necessity for delivering the blood into the vessels. It was just one year later, in 1616, that Harvey described the circulation of the blood.

Who performed the first true blood transfusion from human donor to human recipient remains a matter of doubt. Francisco Folli of Florence claims to have done so in 1654, using a system of cannulas. Christopher Wren and Richard Lower did many experiments transfusing dogs and transfusing animal blood into humans. They may have transfused human blood. Jean Baptist Dennis transfused a man who was near exsanguination from venesection with sheep's blood and he recovered. This was in 1667 and he may have performed other transfusions using human donors. Like many medical discoveries, numerous experimenters were trying to solve the same problems. Within a relatively short period, blood transfusions assumed great popularity and were given a wide trial. The results were disastrous. Infection, air embolism and transfusion reactions, due to improper blood types, resulted in fatalities. It followed that in Rome and France the procedure became illegal and in England it was frowned upon by the Royal Society. For 150 years this was the situation until

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James Blundell, an English obstetrician, used blood transfusions in an effort to overcome the fatalities due to postpartum hemorrhage. He withdrew blood by syringe from a vein to a container from which it was pumped into the recipient by means of another syringe. Again, infection, air embolism, incompatability reactions, the clotting of blood in syringes, and phlebitis were major complications.

The clotting of blood was a problem from the beginning. Bischoff employed defribination of the blood as a means of producing a fluid that would not coagulate and both Larsen and Martin reported successful transfusions using this technic. In addition, artery to vein and vein to vein methods of transfusion continued to be tried with the same unfortunate results, so by 1880 intravenous saline solutions had largely replaced efforts at blood transfusion.

Meanwhile, in the sixteenth century, the Jesuit, Father Kircher, laid the foundation for our modern theories of bacterial infection and Leeuwenhoek, a Dutchman, invented the microscope. Others described the cellular structure of living things and a slow progress was being made which reached its zenith in the discoveries of Louis Pasteur who, by 1880, had made clear the connections between bacteria and disease. The contributors from this point onwards, insofar as infection is concerned, were too numerous to mention. The time was coming when infection of a blood recipient either as a result of failure to use aseptic technic or the use of blood containing a pathogenic organism was to represent a greatly reduced hazard. Serological tests detect syphilis in the prospective donor and other infectious diseases can be kept at a minimum by proper questioning and examination of donors. In countries where malaria is endemic, it is impossible to avoid using donors with a history of malarial infection. Homologous serum jaundice or serum hepatitis remains a problem, as there are many carriers of the virus who give no history of jaundice.

The concern about air embolism has extended from fear of an occasional bubble in the transfusion tubing to relative indifference to the injection of moderate quantities of air. Apparently a few cubic centimeters of air are fatal only if present in a pulmonary vein and the resulting fatality is apparently due to the air bubble entering a coronary artery with resulting myocardial ischemia. The injection of a few cubic centimeters of air into a systemic vein in general seems to be without ill effect. This fact should not justify carelessness in the transfusion procedures. When a considerable amount of air enters a systemic vein death may result from air block of the right ventricle or frothing of the blood with resultant delay in emptying the right ventricle and delayed passage through the pulmonary tree. Occasionally the blood filter of a transfusion set becomes coated and, as a result, the blood level inside the filter is below the level of blood outside. Air may then be trapped in the blood passing through the filter and the tube leading to the recipient will be seen to be full of a mixture of blood and minute air bubbles. A considerable volume of air can enter the systemic circulation if this continues. Obviously the filter should be changed. Occasionally, when a cannula or tube is placed in a vein and the vein wall is not secured tightly around it, air can be drawn into the vein. This condition may exist for a considerable time and produce untoward results. Air leaks in the rubber tubing resulting from puncture with too large a needle while giving an additional injection may permit air to be drawn in if the flow is rapid. This can go on for some time. The amount of air that can be drawn into large needle in the brief space required to change transfusion sets is not large, but even this can be prevented by finger or tourniquet pressure on the vein above. A special hazard exists, of course, when blood is being transfused rapidly under positive pressure from an oxygen tank or hand pump. If the attention of the attendant is distracted for a short period, the bottle may empty and a considerable amount of air may enter the vein in a short period. Even the blood donor may be exposed to danger. If the vacuum is allowed to escape from the present type of tightly sealed vacuum bottle for drawing blood and blood is pumped into the bottle by venous pres-

sure, the air trapped in the bottle will be compressed. There will be air under moderate pressure above the blood in the bottle. If the tourniquet is released from the donor's arm before the needle is removed from the vein, a considerable amount of air can rush into the vein and a sizeable air embolism can result. Fatalities have been reported. It is always a temptation to remove the tourniquet first as the amount of bleeding from the puncture wound is diminished. This procedure is neater but it is more hazardous. The needle should be removed first and then the tourniquet.

In 1900 Landsteiner and Shattuck aided blood transfusions on the road to their present importance by discovering the presence of agglutinating and agglutinable substances in the blood. In 1901 Landsteiner described the existence of three distinct blood types based on these substances, and in 1902 De Costello and Sturli described a fourth. In 1907 Jansky confirmed Landsteiner's work and further confirmation was given by Moss in 1910. Technically such support was desirable and led to rapid acceptance of the existence of such blood types. Unfortunately, these men worked largely independent of each other and adopted different labels for the four basic blood types which created a confusing situation which has not yet been entirely cleared up.

The existence of the four basic blood types rested upon the possible presence in the red blood cells of two specific antigens for which two specific antibodies could be demonstrated in the blood serum. Of these four specific substances no individual has more than two and these must exist in such a distribution that no individual's blood serum contains an antibody capable of agglutinating the antigen carried by his own red cells as that would not be compatible with life. With such axioms, four blood types are possible.

International or Landsteiner	Moss	Jansky	Antigen in Red Cell	Antibody in Serum	Percentage in white races
O	4	1	None	anti-A, anti-B	43
A	2	2	A	anti-B	40
B	3	3	B	anti-A	13
AB	1	4	AB	None	4

In our country use of the International classification is now practically universal.

The reason for severe or fatal blood transfusion reactions became apparent with clarification of these basic blood types. If type A blood is given to a type B individual, the anti-A antibody in the serum of the type B recipient agglutinates the type A red cells of the donor. This is a major mismatch. It is obvious that the anti-B antibody in the donors' serum possesses similar ability to agglutinate the B antigen in the recipients' red cells. In practice, probably due to dilution of the donors' serum in the recipients' serum, this is usually of lesser importance and is referred to as a minor mismatch.

Ottenberg in 1911 pointed out that, inasmuch as the red cell of type O blood carried no antigen, they could not be agglutinated and, therefore, type O blood could be given to any blood type recipient without danger of a major mismatch, so type O became known as the Universal Donor. Similarly, type AB, having no antibodies in the serum capable of agglutinating either the A or B antigen, could be transfused with any type blood without danger of a major mismatch and became known as the Universal Recipient.

It soon became apparent that type O blood was not an entirely safe universal donor blood. The levels of anti-A and anti-B antibodies in the serum of all type O bloods is by no means the same, some have a very much higher titer than others. If the titer is high enough and type O blood is given to a recipient who is not type O, the existing minor mismatch may become serious and the recipients' red blood cells may be agglutinated by the antibodies in the donors' serum, despite its dilution in the recipients' blood stream and a severe or fatal reaction may occur.

For a time the discovery of the Rh antigen, to be discussed later, masked this effect and it was thought that reactions previously reported following use of type O blood, for recipients other than type O, were due to the Rh factor. The work of Aubert, Boorman, Dodd and Loutit in 1942 definitely clarified this point and showed that about 40% of type O individuals had a sufficiently high titer of anti-A antibody

to produce serious reactions. Others confirmed this, although the exact percentages varied, but, in general, about 50% of type O bloods are of low titer and make satisfactory universal donors while the remaining 50% presented some hazard.

Witebsky in 1945 succeeded in preparing group specific substances A and B capable of partially neutralizing the anti-A and anti-B antibodies in the serum of type O donors and the addition of these substances has been practiced on a wide scale in an effort to make all type O blood satisfactory as universal donor blood.

This situation is further complicated by the fact that the anti-A and anti-B antibodies in the blood serum seem to exist in two forms, one of which is the natural antibody producing the usual agglutination of red cells carrying the corresponding antigen, and another called the immune antibody which produced hemolysis of the red cells carrying the corresponding antigen. These immune antibodies are not present universally and are not readily detected by the simpler types of crossmatching technics. They are thought to be produced by pregnancies of different blood type from the mother, by previous transfusions, by recent injection of tetanus antitoxin or toxoid and probably by other unrecognized factors. Neutralization of the immune forms of the anti-A and anti-B antibodies is not readily accomplished by the type specific substances of Witebsky, much greater quantities must be employed and even then complete neutralization may not occur.

When an individual is given blood of an incompatible type, the resulting agglutination, hemolysis or both of the donors' red cells is not necessarily fatal. If the individual recovers he faces an additional hazard. He has had a considerable exposure to a red cell antigen either A or B or both which is not present in his own red cells, although the antibody to that antigen is present in his serum. As a result of this additional exposure the titer of antibody in his serum against that particular antigen may be greatly increased and if at some future time he should again be transfused with red cells containing this same incompatible antigen a rapidly fatal reaction may ensue. At first glance it may seem as if the chance of multiple transfusions with incompatible blood was so remote that it could be deemed unimportant and for most individuals this is certainly true. But it is not true for those who are type A. It is now recognized that the A antigen consists of at least three antigens which are designated as A_1, A_2, and A_3, producing, as a result, three subtypes of type A blood. In addition, type AB blood will actually consist of three subtypes A_1B, A_2B, and A_3B. In typing these bloods, those with the A_1 antigen show rapid and complete agglutinations but when the A_2 or A_3 antigen is present the agglutination may be slow or weak. If the anti A typing sera employed is old or weak, the presence of the A_2 or A_3 antigen may not be recognized. This can result in type A_2 and type A_3, being reported as type O and type A_2B and type A_3B may be reported as type B.

The problem of getting the blood from donor to recipient remained a problem even after blood typing in accordance with the four basic groups had been widely established. The contact of untreated blood with almost any type of foreign material initiated coagulation which might result either in halting the flow of blood or passing coagulated particles on to the recipient. Dr. George Crile devised an artery to vein cannula which brought the intima of the donor artery into contact with the intima of the recipient's vein. This was effective in preventing coagulation but the technic was difficult and required the sacrifice of an artery, two factors which restricted its acceptance. In addition, it could be difficult to judge the volume of blood transfused. The Kimpton Brown paraffin tube was an indirect procedure using unaltered blood as it depended on the smooth surface of the paraffin to delay coagulation. By this technic a known quantity of blood could be transfused. The development of anticoagulants for addition to the blood made indirect transfusions truly feasible. In 1914 Hustin of Belgium carried out indirect transfusions using a glucose sodium citrate solution as an anticoagulant. In the same year Agote, in Buenos Aires, performed a

transfusion using citrate alone. Lewisohn and Weil in 1915 standardized the sodium citrate anticoagulant procedure which has remained basic since that day.

Once it became possible to obtain blood in a fluid state for indirect transfusions, efforts to bank the blood for future use followed. In 1916 Rous and Turner showed that red cells in a glucose citrate solution remained suitable for transfusion as long as four weeks and Robertson in 1917 described 22 cases in which transfusions of one to two liters of blood from 10 to 16 days old was successfully given to American and English troops without serious ill effect.

At the conclusion of World War I little further interest in blood storage was apparent until 1930 when many centers in Russia, Argentina and France reported successful transfusions with bank blood. Study of stored blood showed that the red cells died and underwent hemolysis. This could be delayed by the addition of glucose and storage at low temperatures but the blood would finally become unsatisfactory for transfusion. At that time it was felt blood might be stored satisfactorily as long as thirty to forty days.

The Russians were active in this field. As early as 1865 Sautougin had suggested the use of blood stored at low temperatures for wartime and obstetrical emergencies. By 1937 the Central Institute of Blood Transfusions at Moscow was not only using stored blood on a wide scale but had experimented with the use of blood from cadavers with considerable success. Following the published Russian reports, the first blood bank in this country was established at Cook County Hospital in 1937 by Fantus. Subsequently, blood banks spread throughout the country with great rapidity.

Even though blood can be safely stored and transfused, we need to know something about the fate of its individual components once the blood has been removed from the donor. This problem presents two facets; namely, what happens to blood while it is stored and what happens to either fresh or banked blood after it is transfused. When blood is stored it must be maintained at a temperature between four and ten degrees Centigrade. Lower or higher temperatures, even for a short period, markedly impairs red cell survival time. If the blood is maintained at this temperature, it can be transported by any means without affecting the viability of the cells.

Fresh blood contains a mixture of red cells of varying age. Following transfusion red cells die off in a linear fashion with a maximum life expectancy of about 120 days. After that all the transfused red blood cells are gone. When the blood is stored rather than being immediately transfused, this aging of the red cells continues but is slowed somewhat by the lower temperature of storage. As time passes in storage, some red cells become non-viable, presumably because of derangement of their enzyme systems, some die a natural death, and the survivors all age, even though at a delayed rate, so that when transfused the survival time will always be less than that of fresh blood. Old bank blood therefore has the disadvantage that an appreciably greater volume must be given to obtain any desired increase in circulating red cells. In addition the recipient must dispose of a greater volume of nonviable cells and cellular debris. Because of this, most blood banks have tried to shorten the interval between the taking and giving of blood so as to combine the advantages of instant availability with those of relatively fresh blood. Even though red cells appear intact after storage for several days, they may rapidly disappear from the recipient's blood stream following transfusion having become non-viable. In 1947 Rapaport reported that the use of an acid-citrate-dextrose solution as an anticoagulant for bank blood resulted in a much higher survival of transfused red cells. This observation was verified by many others and the use of the citric acid-trisodium citrate-dextrose or A-C-D solution became standard for banked blood.

Outdated blood is employed for the preparation of blood derivatives. While this is certainly desirable where facilities permit, bank blood does not suddenly become unusable at a particular moment. In a real emergency old blood can be used with con-

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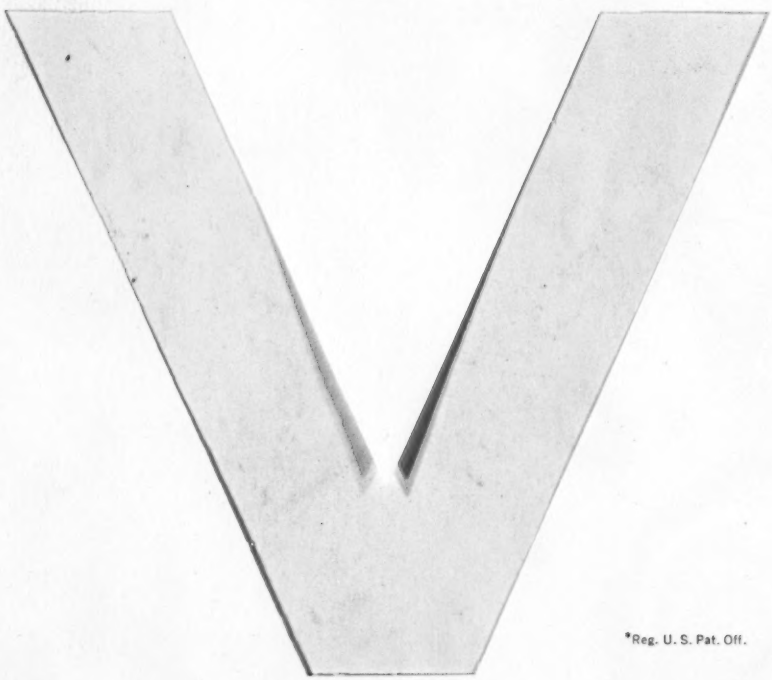

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siderable less hazard than that presented by the emergency. In addition old blood might well be employed for certain cases of chronic anemia or cancer where economic or other factors makes it difficult to obtain fresher blood. Though not the best, it may well be better than none at all.

The problem of bacterial infection arises in connection with the use of old blood. Despite the most meticulous technic, bacteria present in the skin of the donor at the site of puncture may gain entrance to the blood. If the blood is promptly refrigerated and kept so until transfused, no marked growth of the usual pathogenic organisms will occur. However, there are certain organisms, ordinarily not particularly pathogenic, which grow well at the refrigerator temperature for bank blood. Hemolysis and discoloration of the supernatant plasma may not be apparent as the hemolyzed blood fails to break through the upper layer of sedimented white cells. In using older blood, a hanging drop can be examined microscopically for bacteria. This should be done just prior to using the blood for transfusion. It is generally accepted that, unless enough bacteria are present to be visible by this means, a serious reaction will not occur.

When blood is being given over a prolonged period, bacteria can grow in the warm blood that covers the filter with resultant contamination of the blood being transfused. A limit of eight hours should be placed on the use of the same filter.

The National Health Institute sanctions the use of bank blood properly drawn and stored for a period of 21 days from the time of collection. Many blood banks set a limit appreciably shorter than this.

Experiments to determine the life expectancy of white blood cells are difficult to perform. So far as can be judged, it does not exceed a day or two. In blood transfusions, as now performed, it is recognized that the citrate solution is itself toxic to white cells and, in addition, they tend to cling to glass surfaces and become damaged. The white cells do not respond to clumping technics as do the red cells and efforts to trace their survival by such means is of no avail. It can be accepted that when fresh blood is transfused the white cells die very soon and in bank blood stored even an hour or two they are already dead. Present transfusion technics are not adequate for the transfusion of living white cells and, even if satisfactory technics were available, the apparent very short life of the white cell would necessitate retransfusion within a matter of hours, if our object was to maintain an appreciable white cell count in an individual with leukopenia.

The survival of transfused platelets is difficult to determine because of the problem presented in following the transfused platelet after it arrives in the recipient's blood stream. It is thought their survival time does not exceed five days. This is based on transfusion by vessel to vessel anastomosis. When a direct system employing syringes was employed, there were no appreciable platelets transferred to the recipient's circulation. When blood is banked the platelets rapidly disintegrate. Present transfusion technics accomplish no effective transfer of blood platelets. However, in 1945, Jaques, Fidlar, Feldsted, and MacDonald pointed out the importance of protecting blood from contact with the surface of bottles, needles and other apparatus employed in transfusions. Such contact damages the blood cells, platelets, and plasma proteins. Today, silicone preparations are available for the treatment of glass, metal or tubing to produce non-wetable surfaces of either short or long duration as desired. Following this Monor and Burnett have reported on the preparation of platelet concentrates for treatment of individuals with thrombocytopenic purpura, aplastic anemia and acute leukemia. This procedure is of interest because blood drawn into standard transfusion bottles, especially of the siliconized type, containing standard A-C-D solution, can be employed. Using Dextran to speed sedimentation of the formed cells and Triton solution to prevent clumping of the platelets left free in the plasma, a suitable centrifuging technic produces rapid sedimentation of the cells and the platelets dispersed in the plasma can be drawn off.

The plasma component of the blood apparently undergoes no marked alteration in protein content. Present in the plasma are the antibodies that have been developed by exposure to infectious diseases and the use of blood transfusions in the treatment of such diseases rested, in part at least, upon the hope that effective antibodies would be present in the transfused blood. Antibiotics have proven much more effective against bacterial infections and blood transfusions are rarely given for that purpose. However, against viral infections the blood plasma may contain many effective antibodies. It is known that these antibodies are concentrated in the gamma globulin fraction of the blood plasma which is readily separated from the plasma in commercial laboratories and possesses two advantages: the antibodies are concentrated and the virus of serum hepatitis is rarely, if ever, found in gamma globulin, so that its administration is free of this hazard. Antibody molecules have a half life of 14 days but their disintegration follows a logarithmic curve, rather than a linear one, so after two months about 10% of its antibody activity remains. It is apparent that bank blood stored for less than 14 days may have effective antibodies against viral diseases but the concentration will be appreciably less than that to be derived through use of gamma globulin.

With the establishment of blood banks the use of blood transfusions increased at a rapid rate. There were reactions and there were occasional fatalities. Some reactions were due to errors. These were mainly due to the increased work load, haste and clerical errors rather than technical mismatching of blood. There were, in addition, reactions that were not covered by this explanation. The most careful checking of the bloods revealed no mismatch so far as basic type was concerned and attempted explanations based on pyrogenic reactions to foreign material or broken down blood in the apparatus failed to satisfy.

One should not minimize the importance of pyrogenic reactions. Anyone who had occasion to use the old type of transfusion equipment with locally cleaned and resterilized tubing will remember these reactions. The replacement of such equipment with the modern closed disposable transfusion set has made such reactions a rarity.

In 1941 Landsteiner and Weiner reported the discovery of another antigen of the red blood cell which they called the Rh antigen. If this is present in the red cell the blood is called Rh positive; if absent it is Rh negative. About 85% of people are Rh positive and 15% are Rh negative. The Rh antigen is inherited independently of the A and B antigens so there is no connection between the ABO type and the Rh type.

A person who has Rh negative blood may develop anti Rh antibodies due to exposure to Rh positive antigen. This can occur by receiving a transfusion of Rh positive blood. It also may occur when a woman has a fetus with Rh positive blood. This sensitivity takes a few days to develop but once present it is permanent and a hemolytic reaction may occur if that individual is again transfused with Rh positive blood. This Rh factor was the explanation for many of the otherwise inexplicable transfusion reactions which occurred with blood that had been correctly grouped under the ABO system. With the introduction of Rh typing in addition to established ABO typing the incidence of reactions was reduced.

The mechanism by which an Rh positive fetus in the uterus of an Rh negative mother incites the production of immune anti-Rh antibodies in the mother is not known. It may result from fetal red cells crossing the placental barrier, placental infarcts, fragmentation or some other mechanism. It is known, however, that the anti-Rh antibody produced in the Rh negative mother crosses the placental barrier to enter the fetal blood stream. If the titer of anti-Rh antibody in the fetal blood stream is sufficiently high, destruction of the fetal red cells occurs. This condition is known as hemorrhagic disease of the new born or erythroblastosis fetalis. For the mother to possess a sufficiently high titer of anti-Rh

antibody to produce clinical erythroblastosis apparently requires either repeated pregnancies with Rh positive fetuses and a gradually rising titer of anti-Rh antibodies or else a previous transfusion with Rh positive blood.

When these infants are born with severe blood reactions and anemia, the problem of blood transfusions arises. The infant is Rh positive but its own Rh positive red cells are being destroyed by the anti-Rh antibodies received from the mother. To give this infant additional Rh positive red cells is to invite further red cell destruction. However, the infant can be given Rh negative red cells of its own ABO type and these Rh negative red cells will survive for their natural lifetime in the infants circulation, undisturbed by the presence of the anti-Rh antibody. The infant will continue to produce its own Rh positive red cells and these will be destroyed until the anti-Rh antibody obtained from its mother is exhausted. Until this time the infant's condition may necessitate the further transfusion of Rh negative red cells. In general these infants should not be transfused with Rh positive cells until at least two months after birth, at which time it can be assumed that the anti-Rh antibodies are exhausted. The use of adequate transfusions of Rh negative blood will remove the stimulation to the infant's bone marrow to produce red cells which will occur if anemia is present. As these cells would simply be destroyed as produced in severe cases, their production can add to the infant's illness. The transfusion of Rh negative blood into these infants may result in a rapid subsidence of anemia and jaundice.

With this observation as a basis it became apparent that a still greater salvage of seriously ill infants might be accomplished by removing the greater portion of the Rh positive red cells present in the infant at birth and replacing them with Rh negative red cells. It would seem reasonable to believe the hemolytic process might be brought to a halt at once. With the development by Diamond of the technic for catheterization of the umbilical vein it became possible to carry out exchange transfusions.

Some of the early efforts to treat such infants by transfusion of Rh negative blood of proper ABO type resulted in some unfortunate results. In connection with this it was noted by Davidsohn, Levine and others that some Rh negative mothers with high anti-Rh titers failed to produce erythroblastic infants even though the infant was Rh positive. Weiner and Diamond concluded that immune anti-Rh antibodies required an activator present in adult human blood serum which has been called conglutinin. From Witebsky and his group came evidence that the normal new born infant has no appreciable amount of this substance in his blood serum. In those cases where the mother has a high titer of anti-Rh antibody but fails to cause erythroblastosis in an Rh positive fetus there has been an apparent failure of this substance, conglutinin, to enter the fetal circulation from the mother. When sufficient conglutinin is passed to the fetus along with anti-Rh antibodies to produce clinical erythroblastosis it is obvious that in occasional cases either simple transfusions or exchange transfusions with adult whole blood even though of proper ABO type and Rh negative may result in the addition of sufficient conglutinin to the infant's circulation to activate all the anti-Rh antibodies received from the mother and produce the marked increase in red cell destruction and jaundice noticed. In an effort to overcome this, Pennell had recourse to the aspiration of sedimented red cells from the bottom of the bank blood bottle, thereby leaving most of the plasma with its conglutinin behind. For more severe cases the red cells could be washed free of all traces of donor plasma before being transfused.

Approximately 95% of the cases of erythroblastosis fetalis arise from reaction due to the Rh antigen. Of the remaining 5%, an appreciable number are the result of the A and B antigens. These cases almost always occur where a type O mother has a fetus of different ABO type. For some unknown reason the anti-A and anti-B antibodies of the type O mother reach the fetal

circulation in much higher titer than the anti-A antibodies of the type B mother or the anti-B antibodies of the type A mother. This apparently is the reason most of these cases occur with type O mothers.

Determining whether the erythroblastosis of a particular infant is due to the Rh antigen or the A and B antigens may be difficult and time consuming. It is simpler to use type O Rh negative red cells which possess neither the A, B or Rh antigen and can therefore survive in the fetal circulation.

Whenever a special preparation of red cells is made up for transfusion the necessary needling, pipetting or washing introduces the possibility of bacterial contamination. Such preparations should be placed back under refrigeration if not used at once and in any event should be used within 24 hours.

The situation is further complicated by the fact that the Rh antigen has been shown to be not a single pure antigen but to consist of three antigens which have been labelled C, D, and E. Of these, D is the predominant antigen and is the responsible factor in the majority of reactions or cases of erythroblastosis fetalis due to the Rh antigen. Each of these produces a corresponding antibody, namely anti-C, anti-D and anti-E. In present blood grouping technics the anti-D is generally used for the identification of the Rh type and the C and E antigens may react weakly with this. The situation is not one where the blood either reacts strongly Rh positive or else is Rh negative. Instead, considerable skill may be required on the part of the technician to identify the reaction of those giving weak Rh responses so that they will be correctly grouped. When the requisite skill is lacking such blood may be grouped as Rh negative with consequent hazard to any recipient.

Along with the clarification of the A and B antigens and the Rh antigens came the discovery of a large number of other antigens carried by the red blood cells of some individuals. Some of these are common. Many are weak. One must keep in mind that the A and B antigens produce their marked effect when incompatible blood is transfused because the anti-A and anti-B antibodies already exist in the blood serum of a large number of individuals. For the Rh antigen and the large group of other antigens on the red cell, this is not the case. Antibody for these different antigens must be produced in the blood of individuals who are negative for that antigen. For first transfusions in individuals who have never been pregnant, or for first pregnancies in individuals who have never received a blood transfusion there is no significant chance of a reaction based on these antigens.

The antigen systems which may be found in the red blood cell, in addition to the A, B and Rh, are the MNS, KELL, Lutheran, Duffy, Lewis, P, Levay, Jay, Jobbins, GR, and Kidd. Several of these have already been shown to be responsible for deaths occurring from transfusion reactions and erythroblastosis fetalis.

Today with many individuals receiving multiple transfusions, sensitivity to these antigens may become a matter of extreme importance. We must recognize the fact that a patient's blood is highly individual and probably no two are identical. The transfused blood suffices temporarily but, in time, it is destroyed. However, any antigenic substance, carried by the red cells of any of the donor blood that is alien to the recipient, has initiated the production of immune type antibodies for that particular antigen. A single transfusion of compatible ABO type blood may not produce a significantly high titer of these other antibodies but with multiple transfusions or pregnancies this does not remain true.

Skilled technicians using modern blood typing and matching technics will recognize the presence of such antibodies in the serum of the proposed recipient or prospective donor and can reject donors found incompatible. There is another side to the problem. Recipients having a high titer of antibodies to any one or several of these less well known antigens may present a problem insofar as finding a compatible donor is concerned. True, we can screen the incompatible if sufficient care is used, but we may find none who are compatible. With some

antigenic situations, the chances of finding a compatible donor may be on the order of 1 in 10,000 of random donors tested. So within the limits of any one blood bank and its associated technical personnel, it can be readily recognized that such individuals can become essentially untransfusable. Unless a compatible donor is found within a reasonable number of trials, the search becomes technically and economically impossible.

It has been proposed that a National or International Blood Registry be maintained with listings of donors whose blood types are known, not only with regard to our usual ABO and Rh classifications but also with regard to all these other antigens. In time of need a tiring search through a great number of random donors would not be necessary as compatible donors could be determined from the Registry.

When one considers the tremendous number of blood transfusions that have been given during and since World War II, one realizes that this problem, which has been largely one of academic interest in the past, can soon become one of everyday interest to all.

It is of interest that extensive experimental work is being carried out on the quick freezing of blood to very low temperatures and it has been found that red cells can be kept alive for many months. While present technics are elaborate, insofar as the equipment necessary to prepare and freeze the blood or to reconstitute it for transfusion purposes is concerned, yet it seems probable that, in the very near future, reasonably simple means for banking blood for long periods will be available. When this becomes the case, a wide variety of different bloods, which have been completely typed for their antigenic makeup, can be kept available.

DOUBLE PREGNANCY IN A BICORNUATE UTERUS

A REVIEW OF THE LITERATURE WITH A CASE REPORT

EDMUND G. LAIRD, M.D.*, ROGER B. THOMAS, M.D.** and
NOUR E. HALABI, M.D.***
Wilmington, Delaware

Congenital anomalies of the uterus are not mere anatomical curiosities but are potentially serious conditions since it often happens that uterine anomaly is responsible for abortion, miscarriage, premature and difficult delivery, or uterine rupture. Early diagnosis may be necessary and urgent.

ETIOLOGY

Causes of malformations of the female genital tract are not completely known. In early embryonic life the Mullerian ducts give rise to the uterus, both tubes, and the upper two-thirds of the vagina. If this normal sequence is disturbed and fusion of the Mullerian ducts fails to occur completely or in part, the result will be an anomalous development of the uterus or vagina or both. Falls[1] (1939) has expressed the opinion that hormonal failure in intrauterine life might be chiefly responsible for the improper fusion of the two halves of the uterus, as well as for other developmental deficiencies in this organ. On the basis of an observation made by Felix in 1912, Anderson (quoted in Jarcho[2]) believes that many uterine anomalies can be explained on the ground that the left Mullerian duct usually advances slightly before the right and that an error of timing during embryological development might result in a malformation. However Jarcho,[2] Bailey and Miller, DeLee and Greenhill,[3] Moncure[4] and others take a less complicated view. They maintain that various types of uterine anomalies can be attributed to abnormal fusion or lack of fusion, in whole or in part, of the Mullerian ducts.

CLASSIFICATION

Between the premature uterus didelphys and the almost normal uterus arcuatus, a large number of uterine anomalies are possible. We believe that the classification advocated by Jarcho is logical and quite informative.

INCIDENCE AND STATISTICS

There is no definite figure that can be quoted since many of the congenital anomalies of the uterus remain undiagnosed. However, series of observed and collected cases from different sources indicate that the incidence of these anomalies varies from 0.004 to 3.8 per cent.

Fenton and Singh[5] found that uterus didelphys and uterus bicornis unicollis, vagina simplex were the most commonly found abnormalities in their series. During the 25 year period (1925-1949), 62 patients presenting different types of congenital malformations of the uterus and vagina were observed at the obstetric service of the Sloane Hospital for Women. Four of the sixty-two had uteri duplex bicornis bicollis, vagina simplex. In the same 25 year period there were 60,788 deliveries, giving an incidence of 0.00065% of the deliveries. As to the occurrence of uterine abnormalities in pregnancy as a whole, Taylor[6] states that it occurs once in about 1,500 obstetrical and once in about 2,000 gynecologic cases. Eck stated that the duplicity of the genital tract is found most frequently in broadly built women of stable temperament. Falls[1] feels that pregnancy and labor in the arcuate type of bicornuate uterus is of relatively frequent occurrence. Impregnation with normal delivery has taken place with cases of double vagina and double uteri and the condition was discovered later during an operation or on pathological examination of the uterus.

Probably the earliest recorded case of a double uterus associated with pregnancy

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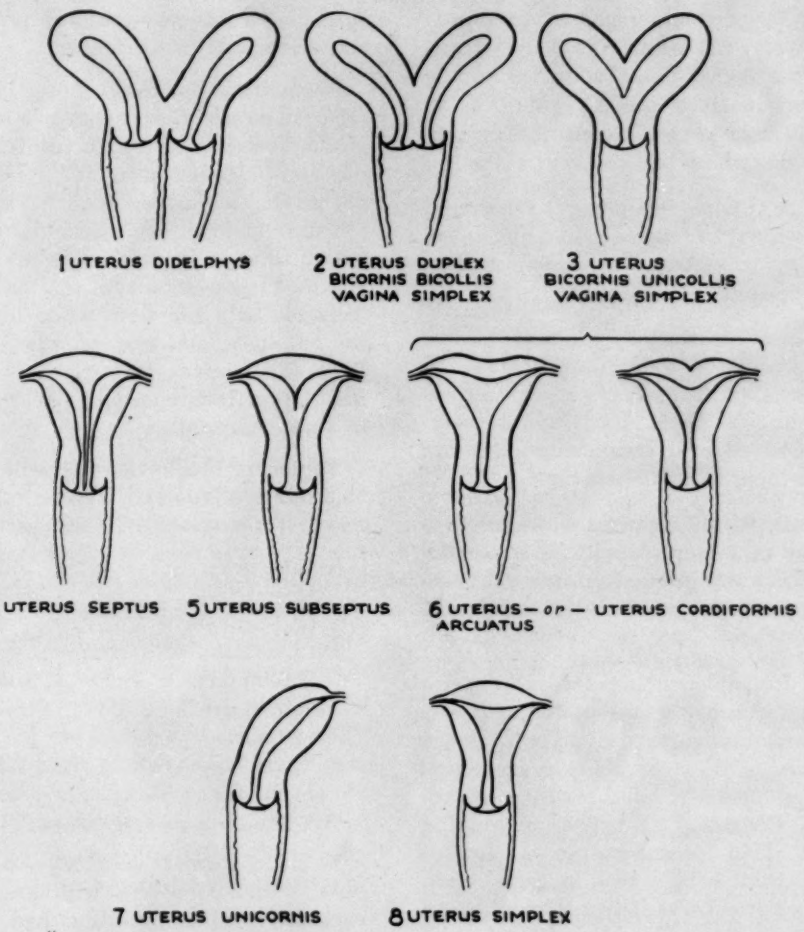
1 UTERUS DIDELPHYS
2 UTERUS DUPLEX BICORNIS BICOLLIS VAGINA SIMPLEX
3 UTERUS BICORNIS UNICOLLIS VAGINA SIMPLEX
4 UTERUS SEPTUS
5 UTERUS SUBSEPTUS
6 UTERUS — OR — UTERUS CORDIFORMIS ARCUATUS
7 UTERUS UNICORNIS
8 UTERUS SIMPLEX

FIG. 1. Normal uterus and seven types of malformation of the uterus and vagina. (Courtesy Jarcho, J.: Malformations of the uterus, Am. J. Surg. 71: 109,1946.

was reported by Mariceau and Vassal in 1669. (quoted in Jarcho[2]). Weise in 1883 reported the first American case. It was followed by a case report by Lane in 1885. In 1900 Wells found fifty-seven pregnancies in 112 cases of double uteri. Boni in 1901 collected 484 single pregnancies in double uteri. Smith[7] studied the records of the New York Lying-In Hospital during a period of 30½ years (January 1899 to July 1930). During this period 35 cases of double uterus were recorded, nineteen of which were observed by Smith personally. He was astonished to note that all types of double uterus had occurred once in 1,458 cases during the five and one-half years of personal observation. Whereas, in the records of 114,243 consecutive patients examined during the previous 25 years, double uterus had been recorded once in 7,040 cases. It was inferred that some cases were not recognized or had not been recorded.

In Germany, Pfleiderer[8] reported that, during a period of 20 years, 93 instances of genital anomaly were observed at the Tubingen Frauenklinik. Fourteen were of rudimentary solid uterus with solid vagina, six of uterus unicornis with rudimentary horn, fifteen of uterus bicornis unicollis, sixteen of uterus septus with double or single vagina, and twenty-seven of uterus arcuatus.

In their report on pregnancy in uterus duplex, Acosta-Sison and Katigback[9] stated that of the 68,682 cases admitted to the Philippine General Hospital from 1912 to 1937, only 3 cases of bicornate uterus were noted.

Moncure (quoted in Corbet[10]) reported a case in which two babies in a didelphys uterus were delivered by a Cesarean section. They were easily resuscitated.

In a case reported by Corbet,[10] a live fetus was delivered spontaneously from the right uterus and three days later a second living child was born. Each uterine component behaved as an independent unit and went into labor at a different time.

Bhagwat, S. A.[11] reported a case of twin pregnancy in a uterus duplex in which the two labours took place at an interval of 26 days. Both babies were alive at the time of each delivery.

David Rosenbloom[12] reported a full term pregnancy in a bicornuate uterus with premature separation of central placenta previa which was delivered by a Cesarean section because of uncontrollable bleeding on admission. Charles J. Kickham reported a full-term twin pregnancy in a "uterus septus". Both babies were in good condition. The patient was delivered by a Cesarean section because, on admission, she was in a toxaemic condition. On the eleventh postoperative day, the patient had a typical uraemic type of convulsion and died in about one hour. No permission for autopsy was obtained.

Arthur B. Barrett[13] reported a case of bicornate uterus with pregnancy in each horn. The patient was admitted with threatened premature labor, and delivered one baby spontaneously with the aid of low forceps without difficulty. Five minutes later the placenta was expressed from the left horn. The left horn did not contract well and more than usual bleeding continued. For this reason delivery of the right horn was done by internal podalic version and extraction. Postpartum recovery was uneventful. Barrett also reported in 1934 a bicornuate uterus with a pregnancy in each horn. Both infants were born prematurely and died shortly after delivery.

Hagood and Eastluck (quoted in Wong[15]) reported a case of pregnancy in a bicornuate uterus complicated by an incarceration of the non-pregnant horn. The details were not given. Jellinghaus reported a case of a full term child in one uterine horn and a 4 month fetus in the other. He also reported another instance where the patient was delivered of a full term white infant from the left horn, and two months later a full term black infant from the right horn of the uterus. Ross reported a triple pregnancy in a double uterus.

Taylor[6] in 1943 reviewed the literature on bicornuate uterus and found a total of 271 cases. He summarized them as follows:

Total cases	271
Obstetric	265
Gynecologic	6
Incarceration of a nonpregnant horn	12 or 4.5%
Maternal death	4 or 1.5%
Abortions:	
Smith	12.8%
Schauffler	53.0%
Average, DeLee and others	25.9%

In 1925 Rowlett of Florida described a case of double uterus with pregnancy of each horn. The condition had been discovered during a laparotomy two years previously. Despite this knowledge the patient went into labor after five months' gestation, miscarrying male fetuses weighing 3 pounds 4 ounces and 2 pounds 3 ounces. The question of superfetation was raised.

In 1933, Wong[15] of the Peiping Union Medical College reported on a pregnancy in one horn of a double uterus. At six months, there occurred all the symptoms of a threatened miscarriage resulting in expulsion of a decidual cast of the nonpregnant horn. Three and one-half months later the patient was delivered of a normal girl baby.

In 1939, Johnston of Akron, Ohio, reported a case with a dead fetus in one horn and a normal pregnancy in the other. The condition was discovered during a laparoto-

(Continued on Page 82)

Editorial

The tremendous advances in medicine and the trend to specialization have placed the general practitioner in a most important position — the first line of defense. In war it is the infantry, in football the linemen and in medicine the family doctor whose actions make the difference between success or failure.

PREVENTION OF DEFORMITY

Physical Medicine and Rehabilitation is a relatively young but none the less important medical specialty. In another part of this Journal there is an article in which the author pleads with the general practitioner to carry out certain simple procedures in caring for the patient who has had a cerebral accident. These simple procedures are commonly used in the large teaching centers and have been proven effective in the prevention of residual deformities. In private practice, most patients with hemiplegia are treated by the general practitioner. It therefore is his responsibility to adopt any safe and simple measures that have been shown to be effective in treating these patients.

MENTAL ILLNESS

Psychiatry is an old and time honored medical specialty. It is the opinion of authorities, however, that the advent of the new tranquilizing drugs has created a situation in which the family physician is prescribing more medication for the mentally disturbed individual than is the psychiatrist. This may be a good thing because in his work at the front line the family doctor sees mental illness in its earliest stages and is in a position to bring psychiatry to all segments of the population. This move of psychiatry from the mental hospital into the community is a good thing. It raises the problem, however, of post-graduate training in psychiatry for the family doctor. The American Academy of General Practice already has declared that the general practitioner desires this training. The National Mental Health Committe is now proposing that the Congress allocate funds for such post-graduate training. Their proposal is realistic inasmuch as the general practitioner would be compensated on a scale in proportion to his experience and obtaining such training would not entail too great a financial sacrifice. This is indeed a step in the right direction.

bicornuate uterus with a pregnancy in each

(Continued on Page 82)

(Continued from Page 80)

my in which the cornu with the dead fetus was mistaken for a fibroma and removed. The remaining pregnancy continued uneventfully to term.

In 1943, Major Braze[16], M.C., U.S. Army, reported a case of pregnancy in each horn of a bicornuate uterus which proceeded uneventfully to term with delivery of two normal viable children.

The following is a case report of a woman with a uterus duplex bicornis bicollis, vagina simplex who was first recognized as having a bicornuate uterus at the age of 36 years. She was the mother of one child 11 years old and another 7 years old. All indications lead to the assumption that her second conception was a double pregnancy resulting in a miscarriage at four months and a normal, living, female child at term.

CASE REPORT

A 36 year old white female had a spontaneous miscarriage on July 8, 1946. Her last menstrual period had been on April 1, 1946. The fetus was inspected personally by one of the authors at the patient's home. The patient continued to bleed for a short while thereafter but made an uneventful recovery.

At the time of her miscarriage, she gave a history of having had regular menstrual periods at 26 day intervals but had always suffered from mittelschmerz and a slight, bloody vaginal discharge at such times. She had had one healthy child on February 27, 1943, and her last menstrual period before this delivery was on June 16, 1942, i.e. approximately eight months previously.

On January 4, 1947, six months after her miscarriage, she was delivered spontaneously of a full term female baby. A short time thereafter a posterior colporrhaphy was performed by another surgeon. No abnormalities of the uterus were noted at this time.

On November 13, 1954, she was examined by one of the authors because of intermenstrual bleeding and a profuse leukorrhea. Pelvic examination showed a marital introitus with a well performed posterior vaginal repair. There was no vaginal septum and only one cervical os could be seen. There was a chronic endocervicitis with moderate erosion. The uterus was found to be unequivocally bicornuate and both cornua were large, symmetrical and of approximately equal size. The cervix was thick when compared with the size of the horns and quite hard. Both ovaries were palpable and of normal size. No adnexal masses were felt. A dilatation and curettage with cauterization of the cervix was recommended.

Operation (Dec. 13, 1954): The cervix revealed a moderate erosion and a chronic endocervicitis. No septum of the external os could be seen but, by means of a uterine probe, a septum was found to be present which extended down to just within the external os, dividing the lower uterine segment into two equal compartments. Both uterine cavities were symmetrical and approximately equal in size, although the probe passed consistently more readily into the right uterine horn. Each side measured three and one half inches in depth. The curettings were normal in appearance and the subsequent pathological report was: "Late secretory endometrium".

Following the operation the patient continued to have intermenstrual spotting.

On October 22, 1955 the patient was again examined by one of us because of upper abdominal discomfort. A cholecystogram on November 30 revealed partially calcified stones in the gallbladder.

Operation (January 16, 1956): A right paramedian incision was made. The gallbladder contained two stones and was removed. Exploration of the pelvic organs substantiated again that the patient had a bicornuate uterus. Both of the cornua were of approximately the same size and were symmetrical, but the left cornua was perhaps slightly smaller than the right. The lower uterine segment felt thick and indurated as compared with the cornua. Both ovaries felt normal.

SUMMARY

1. A brief review of the reported cases of double pregnancy associated with bicornuate uterus has been presented.

2. An additional case is reported in which a woman with a proven uterus duplex bicornis, vagina simplex had a double, simultaneous impregnation which resulted in a spontaneous miscarriage at three months followed by a spontaneous delivery of a normal, female child at nine months.

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A LETTER FROM DR. EDGAR MILLER

CONTINUED FROM FEBRUARY ISSUE

systolic and 50 - 60 diastolic are the common recordings. However, one case we have on the ward now had an admitting blood pressure of 220/110. The present pressures average 150/90, after having received Apresoline and Serpasil. The background here is renal according to his Fishberg test and urinary findings. Unfortunately, at this time we do not have the material to do the Regitine test. His fundoscopic examination reveals first degree retinal arteriosclerosis. His electrocardiogram shows the inverted T waves with coved-shaped S-T segments as seen in long standing hypertension.

Autopsies are prohibited except in criminal cases. Fortunately we have had no such cases. Recently I saw over a thousand people gathered around an autopsy room in the Government Hospital concerning a case of questionable drowning. It took three policemen to control the crowd and the three doctors on the board had to be protected two days afterwards. As a patient is about to die they generally rush him out of the hospital and carry him to the banks of the sacred Baghmati River which is a branch of the Ganges. If they cannot make the river they must die on the earth. Five o'clock the other morning one of our heart cases suddenly died, and due to the lack of help at that hour I helped to carry him out of the hospital to Mother Earth to breath his last. The body is then carried to the river and cremated. One often sees the arms or feet protruding from a pyre.

Incidentally our electrocardiograph machine is working fine. We have taken over 70 tracings thus far. This is the first instrument in the country. Heretofore a machine would be flown up from India, as in the case of the late King Tribiuana. Recently one of our best physicians, the physician head of the military, or as in our country "The Surgeon General," had a typical massive anterior infarction as shown pathognomonically by the electrocardiograph and unfortunately he expired the next day. At least our Mission Hospital could give a diagnostic report to the attending Nepalese physician. This physician is one of their best, well cultured, well versed, and a very keen diagnostician as well as therapeutist.

The work in general is very rewarding as well as frustrating. There are so many fevers of undetermined origin, so many complaints of "Pate man duckhsa" (pain in the stomach), so much undernutrition, so much tuberculosis of all types including Pott's Disease in children, so much leprosy, and various and sundry diseases — not to mention maimed, crippled, blind, and deaf. There is goitre, hernia, hydrocele, malaria, and elephantiasis from filiariasis. How we long for a specialist in every field! The Nepalese people seem to appreciate our efforts; at least they have been most kind. One exception is the Communist paper which published a fictitious report that of twelve patients we operated on eleven died. Also, a woman was said to be blinded by medicine, and we tied patients down and tortured them. A week later His Majesty the King with his Queen suddenly appeared and paid us a visit for over an hour and viewed our hospital. Furthermore, they accepted our invitation to participate in the official opening ceremonies of our hospital in November.

We feel we can do a better job after our equipment arrives, fortunately most is coming in now after getting through the red tape of import licenses and customs. We feel so inadequate with the lack of personnel, lack of tools, and lack of medicine. After two and a half decades of practicing medicine under ideal conditions, this requires quite an adjustment.

For the time I must close. To describe the ritual of a consultation, or attending the clinics, or making rounds through the wards, or even the problem of prescription writing would require a letter. If you are interested in another four months or so from now we would like to tell you of our progress.

Yours sincerely,
EDGAR R. MILLER, M.D.

October 1956
Shanta Bwahan
Kathmandu, Nepal

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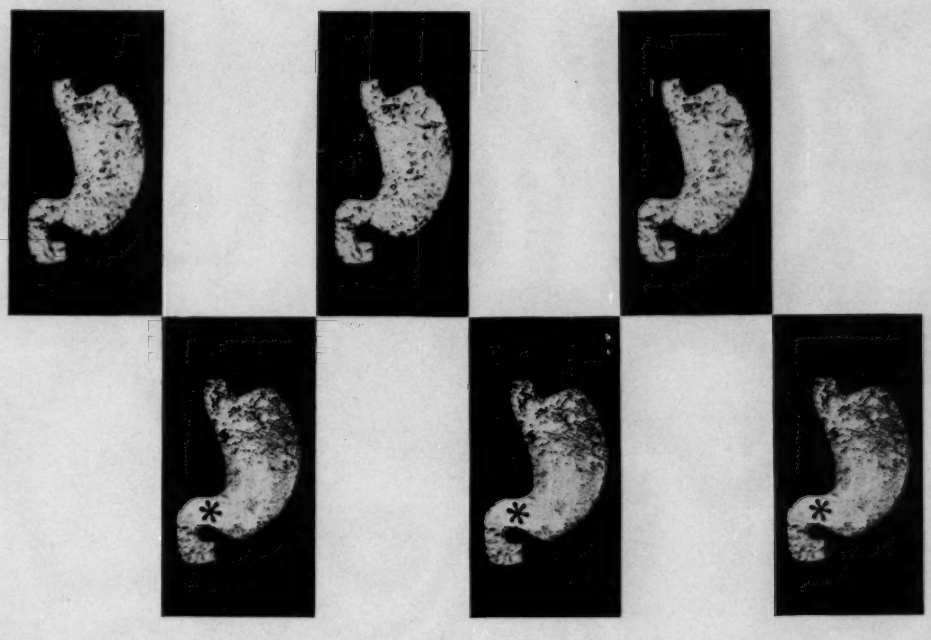

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SURGICAL BELTS
ELASTIC STOCKINGS
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EVERY WOMAN WHO SUFFERS IN THE MENOPAUSE DESERVES "PREMARIN"®

widely used natural, oral estrogen

AYERST LABORATORIES
New York, N. Y. • Montreal, Canada
5645

NOW—EFFECTIVE STEROID HORMONE THERAPY OF RHEUMATIC AFFECTIONS WITH GREATER SAFETY AND ECONOMY

PABALATE'-HC

Robins

Pabalate with Hydrocortisone

Clinical evidence indicates that, in Pabalate-HC, the synergistic antirheumatoid effects of hydrocortisone, salicylate, para-aminobenzoate, and ascorbic acid achieve satisfactory remission of symptoms in *up to 85% of cases studied*

—with a much higher degree of safety

—even when therapy is maintained for long periods

—at significant economy for the patient

Each tablet of Pabalate-HC contains 2.5 mg. of hydrocortisone—50% more potent than cortisone, yet not more toxic.

FORMULA

In each tablet:

Hydrocortisone (alcohol)	2.5 mg.
Potassium salicylate	0.3 Gm.
Potassium para-aminobenzoate	0.3 Gm.
Ascorbic acid	50.0 mg.

DOSAGE: Two tablets four times daily. Additional information on request.

AVAILABLE FOR YOUR PRESCRIPTION NOW

A. H. ROBINS CO., INC. RICHMOND 20, VIRGINIA

Ethical Pharmaceuticals of Merit since 1878

added certainty in antibiotic therapy

–particularly for that 90% of the patient population treated in home or office where sensitivity testing may not be practical . . .

for your entire patient population

multi-spectrum

Sigmam

100% EFFECTIVE in respiratory infections including the 25% due to resistant staphylococci.[1-3]

97% EFFECTIVE in dermatologic and mixed soft tissue infections including the 22% resistant to one or more antibiotics.[3-6]

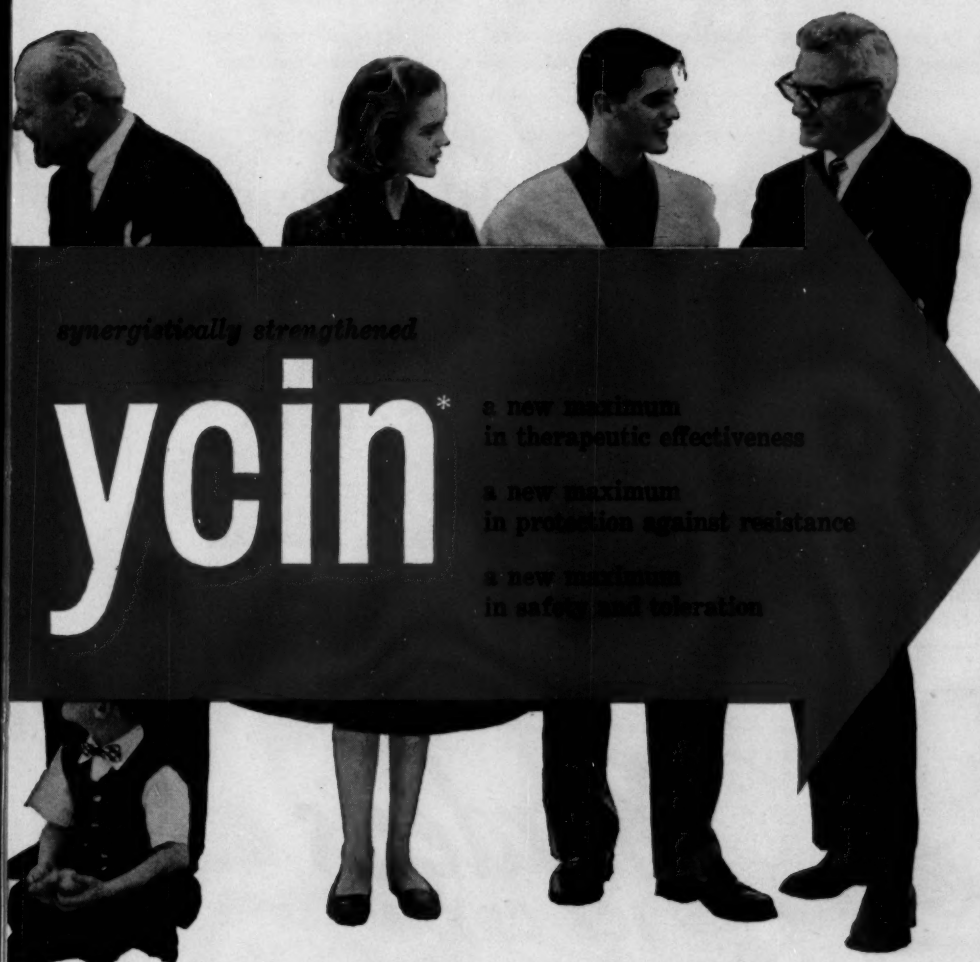
84.6% EFFECTIVE in genitourinary infections including the 61% resistant to other antibiotic therapy.[2,5]

93% EFFECTIVE in diverse infections including the 21% due to resistant pathogens.[1,5]

98.7% EFFECTIVE in tropical infections including those complicated by heavy bacterial contamination or multiple parasitisms.[7]

1. Carter, C. H., and Maley, M. C.: Antibiotics Annual 1956-1957, New York, Medical Encyclopedia, Inc., 1957, p. 51. 2. Shalowitz, M., and Sarnoff, H. S.: Personal communication. 3. Shubin, M.: Personal communication. 4. La Caille, R. A., and Prigot, A.: Antibiotics Annual 1956-1957, New York, Medical Encyclopedia, Inc., 1957, p. 67. 5. Winton, S. S., and Cheserow, E.: Antibiotics Annual 1956-1957, New York, Medical Encyclopedia, Inc., 1957, p. 55. 6. Cornbleet, T.: Personal communication. 7. Loughlin, E. H.; Mullin, W. G.; Aleinder, L., and Joseph, A. A.: Antibiotics Annual 1956-1957, New York, Medical Encyclopedia, Inc., 1957, p. 63.

†the antimicrobial spectrum of tetracycline extended and potentiated with oleandomycin (Matromycin®) to combat resistant strains of pathogens—particularly resistant staphylococci—and to delay or prevent the emergence of new antibiotic-resistant strains.



synergistically strengthened

ycin*

a new maximum
in therapeutic effectiveness

a new maximum
in protection against resistance

a new maximum
in safety and toleration

SUPPLY
CAPSULES: 250 mg. (oleandomycin 83 mg., tetracycline 167 mg.). Bottles of 16 and 100.
new mint-flavored ORAL SUSPENSION: 1.5 Gm., 125 mg. per 5 cc. teaspoonful (oleandomycin 42 mg., tetracycline 83 mg.) 2 oz. bottle.

*TRADEMARK

Pfizer

PFIZER LABORATORIES, *Division, Chas. Pfizer & Co., Inc.*, Brooklyn 6, N. Y.
World leader in antibiotic development and production

ADEQUATE WIRING Certified
REG. U. S. PAT. OFF.

Make Sure You Have Enough

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"Housepower" is adequate wiring . . . enough electrical power coming into your home to meet the needs of all your electrical uses . . . sufficient number of circuits, outlets and switches for your convenience . . . large enough wires so that the flow of electricity to your appliances is not "choked off."

Be sure your home has adequate "Housepower" for all the appliances you want to use today—and in the future. Adequate wiring for today's modern electrical living . . . serves and saves.

Ask your Electrical Contractor to check your HOUSEPOWER today!

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Quality Minded Folks
Who Are Thrift Conscious

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George T. Tobin & Sons
BUTCHERS

NEW CASTLE, DELAWARE
Phone N. C. 3411

Borden's
ICE CREAM

Meat Protein...

and the Many Physiologic Functions of Its Amino Acids

The amino acids supplied by meat protein function in many vital ways in addition to their well-known role in the growth and maintenance of tissues. They participate in the body economy as precursors of hormones, vitamins, enzymes, and other physiologic agents.*

Some of the important amino acids supplied by the protein of meat include: tryptophan (utilized for the endogenous production of niacin); tyrosine (the precursor of thyroxine and triiodothyronine); phenylalanine (converted to melanin, a pigment found in the skin, hair, retina, and other tissues; both phenylalanine and tyrosine are precursors of the hormones noradrenalin and adrenalin); glycine (participates in the formation of glutathione, a tripeptide important in tissue oxidation, in the biosynthesis of glycocholic acid, and in the production of purines, uric acid, and porphyrins used structurally for hemoglobin, cytochromes, and iron-containing enzymes); methionine (an important lipotropic agent; participates in transmethylation processes in which creatine, adrenalin, and choline phospholipids are formed).

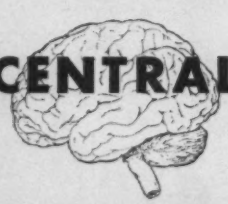
Top quality protein, as supplied by meat, yields important amino acids for participation in these and other important functions. The excellent balance of available amino acids is an outstanding feature of meat protein.

*Geiger, E.: Digestion, Absorption and Metabolism of Protein, in Wohl, M. G., and Goodhart, R. S.: Modern Nutrition in Health and Disease, Philadelphia, Lea & Febiger, 1955, pp. 98-143.

The nutritional statements made in this advertisement have been reviewed by the Council on Foods and Nutrition of the American Medical Association and found consistent with current authoritative medical opinion.

American Meat Institute
Main Office, Chicago... Members Throughout the United States

Both CENTRAL and PERIPHERAL control of cough



SYNEPHRICOL® cough syrup

ANTITUSSIVE • DECONGESTANT • ANTIHISTAMINIC

Combines:
Central Antitussive Effect — mild, dependable
Topical Decongestion — prompt, prolonged
plus Antihistaminic and Expectorant Action

Winthrop LABORATORIES
NEW YORK 18, N. Y.

Each teaspoonful (4 cc.) contains:

Neo-Synephrine® hydrochloride	5.0 mg.
Thenfadil® hydrochloride	4.0 mg.
Dihydrocodeinone bitartrate	1.33 mg.
Potassium guaiacol sulfonate	70.0 mg.
Ammonium chloride	70.0 mg.
Menthol	1.0 mg.
Chloroform	0.02 cc.
Alcohol	8%

Bottles of 16 fl. oz.

EXEMPT NARCOTIC

PHENAPHEN® PLUS

NOSE COLD

each coated tablet:

Phenacetin (3 gr.)	194.0 mg.
Acetylsalicylic Acid (2½ gr.)	162.0 mg.
Phenobarbital (¼ gr.)	16.2 mg.
Hyoscyamine Sulfate	0.031 mg.
Prophenpyridamine Maleate	12.5 mg.
Phenylephrine Hydrochloride	10.0 mg.

Robins

PARKE

Institutional Supplier Of Fine Foods

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COFFEE TEAS
SPICES CANNED FOODS
FLAVORING EXTRACTS

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L. H. Parke Company
Philadelphia - Pittsburgh
7746 Dungan Rd., Phila. 11, Pa.

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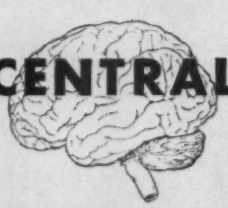

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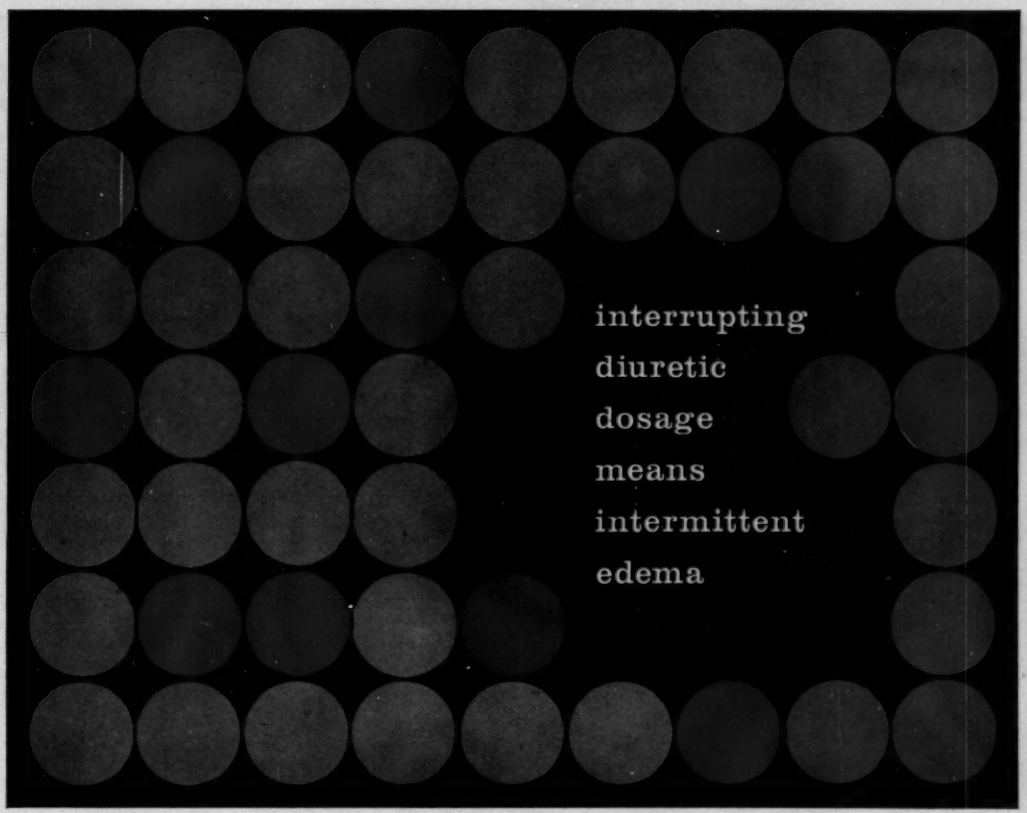


PATIENTS IN FAILURE NEED AN ORGANOMERCURIAL

Diuretics needing "rest periods," whether enforced by dosage restriction to once daily, or by omission to alternate days, inevitably fail to achieve sustained control of edema.

The organomercurials never require interruption of dosage to prevent refractoriness and can maintain patients continuously in the edema-free state.

TABLET

NEOHYDRIN

BRAND OF CHLORMERODRIN (19.3 MG. OF 3-CHLOROMERCURI-2-METHOXY-PROPYLUREA EQUIVALENT TO 10 MG. OF NON-IONIC MERCURY IN EACH TABLET)

a standard for initial control of severe failure

MERCUHYDRIN® SODIUM
BRAND OF MERALLURIDE INJECTION

LAKESIDE

02358

For the Prescribed Diet

LOW SODIUM BREAD

developed by RICE

Specially formulated to reduce the calorie and sodium content, yet provide the nutritive essentials of enriched white bread. Analyses by three independent research laboratories confirmed these specifications:

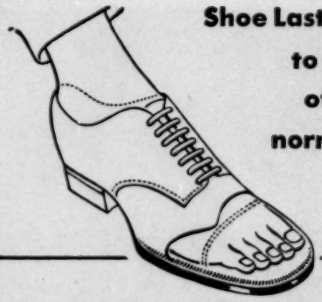
.42 *milligrams of sodium per 18.8 gram slice*

47 *calories per slice*

LOW SODIUM BREAD, double-wrapped in aluminum foil and waxed paper, is delivered to the home by Rice routemen and is available at all Rice Bake Shops. For home delivery telephone MUlberry 5-6800 in Baltimore; OLympia 2-1043 in Wilmington.

RICE'S BAKERY

Baltimore • Wilmington



Shoe Last designed to the shape of average normal foot★

- Insole extension and wedge at inner corner of heel where support is most needed.
- The patented arch support construction is guaranteed not to break down.
- Innersoles guaranteed not to crack or collapse.
- ★ Foot-so-Port lasts designed and the shoe construction engineered with orthopedic advice.
- Conductive Shoes for surgical and operating room personnel. N.B.F.U. specifications.
- We are also the manufacturer of the Gear-Action Shoe designed by noted orthopedic surgeon.
- We make more shoes for polio, club feet and disabled feet than any other shoe manufacturer.

Send for free booklet, "The Preservation of the Function of the Foot Balancing and Synchronizing the Shoe with the Foot."

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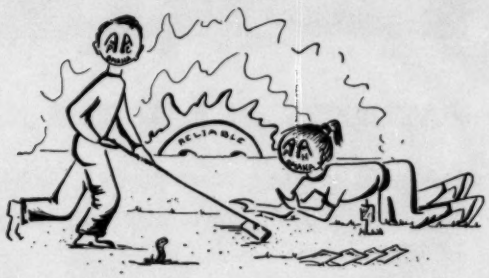
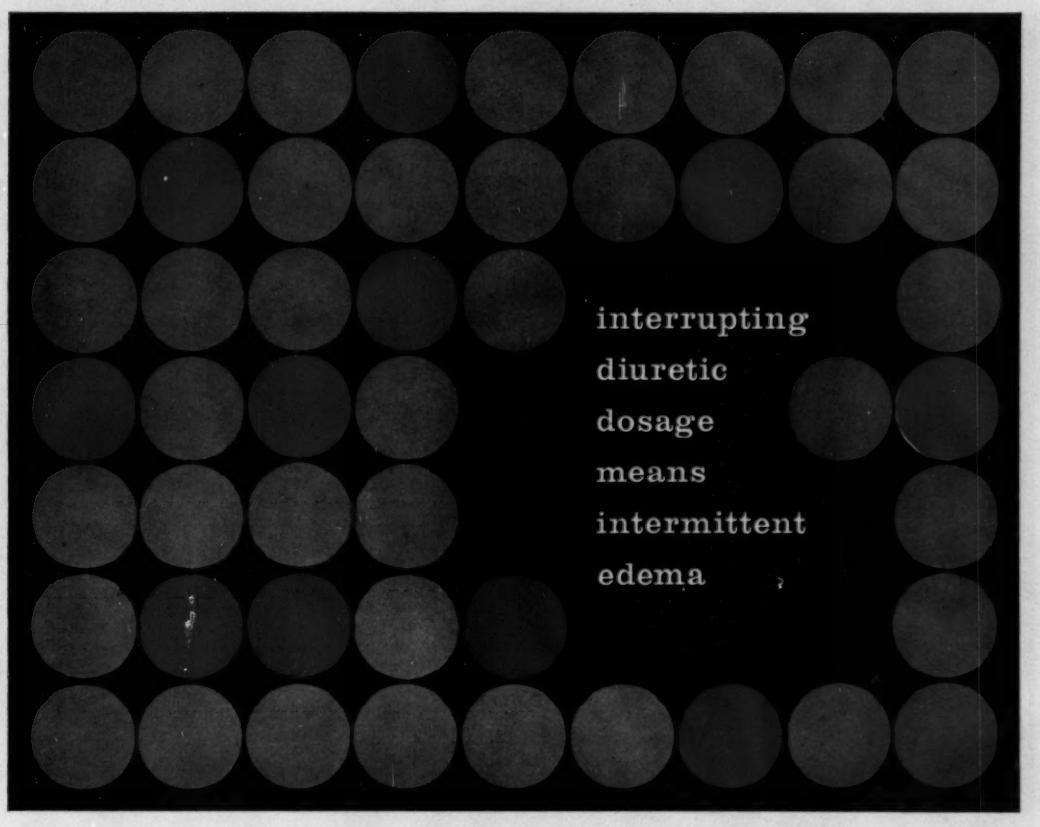
Foot-so-Port Shoe Company, Oconomowoc, Wis.
A Division of Musebeck Shoe Company



PROTECTION AGAINST LOSS OF INCOME FROM ACCIDENT & SICKNESS AS WELL AS HOSPITAL EXPENSE BENEFITS FOR YOU AND ALL YOUR ELIGIBLE DEPENDENTS.

ALL PREMIUMS COME FROM — PHYSICIANS SURGEONS DENTISTS — ALL BENEFITS GO TO

PHYSICIANS CASUALTY & HEALTH ASSOCIATIONS
OMAHA 2, NEBRASKA
Since 1902



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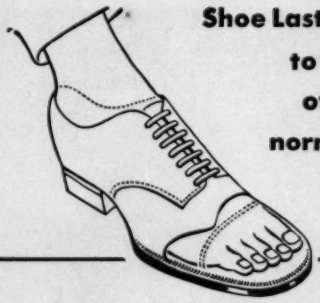
RICE'S BAKERY

Baltimore • Wilmington



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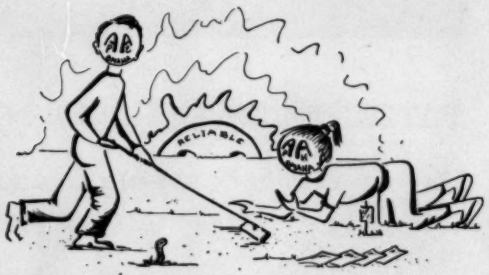

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relaxes both mind and muscle

for anxiety and tension in everyday practice

- nonaddictive, well tolerated, relatively nontoxic
- well suited for prolonged therapy
- no blood dyscrasias, liver toxicity, Parkinson-like syndrome or nasal stuffiness
- chemically unrelated to chlorpromazine or reserpine
- does not produce significant depression
- orally effective within 30 minutes for a period of 6 hours

Indications: **anxiety and tension states, muscle spasm.**

THE ORIGINAL MEPROBAMATE

Miltown®

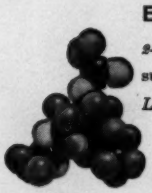
Tranquilizer with muscle-relaxant action

DISCOVERED AND INTRODUCED BY WALLACE LABORATORIES, *New Brunswick, N. J.*

2-methyl-2-n-propyl-1,3-propanediol dicarbamate—U. S. Patent 2,724,720

SUPPLIED: *400 mg. scored tablets. Usual dose: 1 or 2 tablets t.i.d.*

Literature and Samples Available on Request



THE MILTOWN ® MEPROBAMATE MOLECULE

CM-3706-R3

For the Prescribed Diet

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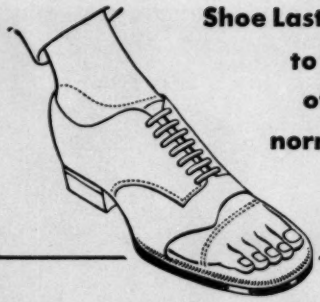

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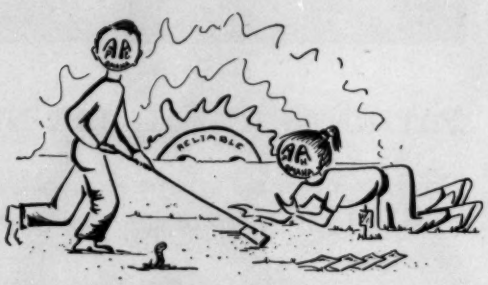

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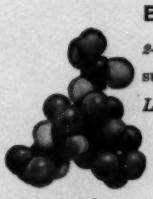

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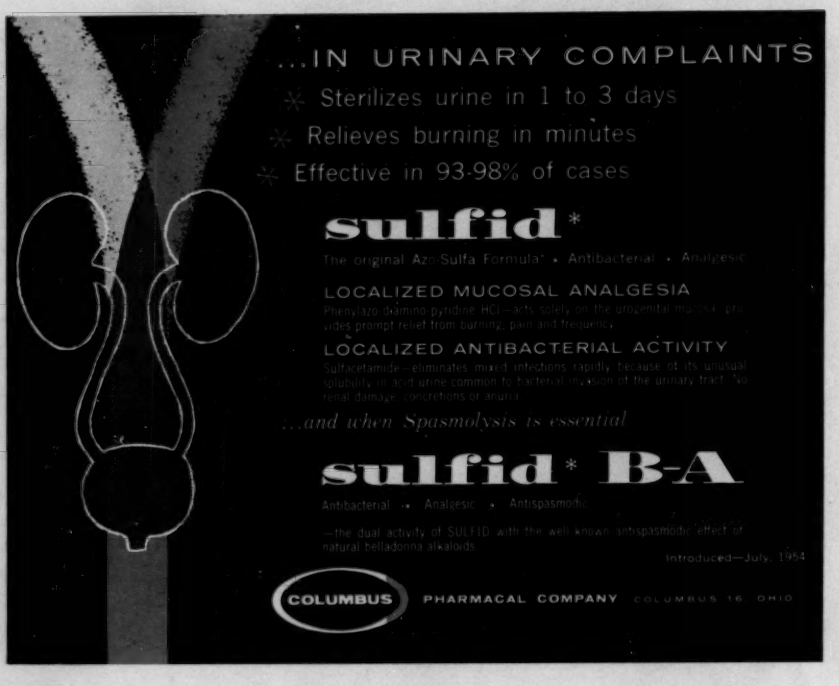

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Quality Dairy Products Since 1900

GOLDEN GUERNSEY MILK

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Flowers . . .

Geo. Carson Boyd

at 216 West 10th Street

Phone 8-4388

outstanding appetite stimulant

INCREMIN*

LYSINE-VITAMIN SUPPLEMENT LEDERLE

Problem-eaters, the underweight, and generally below-par patients of all ages respond to INCREMIN.

INCREMIN offers l-Lysine for protein utilization, and essential vitamins noted for outstanding ability to stimulate appetite, overcome anorexia.

Specify INCREMIN in either Drops (cherry flavor) or Tablets (caramel flavor). Same formula. Tablets, highly palatable, may be orally dissolved, chewed, or swallowed. Drops, delicious, may be mixed with milk, milk formula, or other liquid; offered in 15 cc. polyethylene dropper bottle.

Each INCREMIN Tablet
or each cc. of INCREMIN Drops *contains:*

l-Lysine	300 mg.	Pyridoxine (B_6)	5 mg.
Vitamin B_{12}	25 mcgm.	(INCREMIN Drops contain 1% alcohol)	
Thiamine (B_1)	10 mg.		

Reg. U. S. Pat. Off.

Dosage only 1 INCREMIN TABLET or 10-20 INCREMIN Drops daily.

Lederle

LEDERLE LABORATORIES DIVISION
AMERICAN CYANAMID COMPANY
PEARL RIVER, NEW YORK

Darlington Sanitarium

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(Route 842)
WEST CHESTER,
PENNSYLVANIA

A recognized private psychiatric hospital. Complete modern diagnostic and therapeutic services. Qualified physicians may retain supervision of patients.

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1. Locket, S.: Brit. M.J. 1:809 (Apr. 2) 1955.

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2. Wright, W.T., Jr., et al.: J. Kansas M. Soc. 57:410 (July) 1956.

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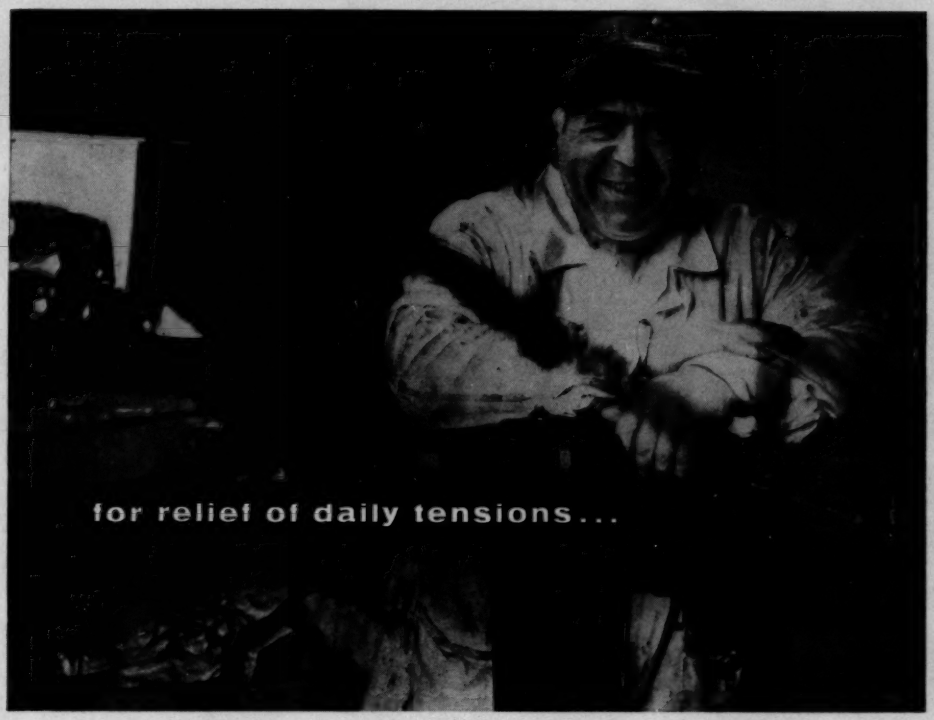

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*Ferguson, J. T.: J. Am. Geriatrics Soc. *4*:1080, 1956.

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1. Personal communication to S.K.F.

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